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ORIGINAL COMMUNICATIONS.

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ORAL COMPLICATIONS OF THE EXANTHEMATA.*

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At the Ninth International Otological Congress in August, 1912, the writer presented a paper based upon a study of the aural conditions found in 454 autopsies after death from diphtheria, scarlet fever and measles. The following paper is practically a continuation of that article and is based largely upon the figures and facts given at that time. The writer wishes to state further that what he is about to say refers entirely to aural complications of the exanthemata and not to aural diseases in general. Also, that the symptoms and treatment given refer only to severe cases often seen in hospital practice and not to the mild cases which are the variety usually seen in private practice.

Writers upon this subject state that aural complications in the exanthemata occur in from 5 to 44 per cent of the clinical cases, depending upon the particular disease present. In the fatal cases we find this percentage greatly increased. In the autopsy reports previously mentioned, it was 82 per cent in diphtheria, 94 per cent in scarlet fever, and 100 per cent in measles. Thus we find aural complications to be very decidedly important factor in this special class of diseases.

We should naturally expect unrecognized and untreated middle-ear inflammation or mastoiditis to go on to meningitis, brain

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abscess and jugular thrombosis. In this respect the autopsy records are disappointing. There were but four cases of septic meningitis (not necessarily of middle-ear origin), but one case of infection of the jugular vein and not a single brain abscess. Judging from this extremely small number of serious complications, acute middle-ear infection and mastoiditis are not as important as we have supposed them to be.

If meningitis, brain abscess and jugular thrombosis do not often occur from diseased conditions of the aural cavities, complications in the heart, pleura, joints and kidneys co-exist with startling frequency. Is there any evidence to prove that inflammation in one organ is in any way dependent upon infection in another? The position of the tonsil is well established in this respect and we know it is frequently the source of infection in other organs. Why, then, may not the middle ear occupy exactly the same position and be the primary source of infection in many of the complications in the various organs which are so frequently affected? To establish such a theory we need only to turn to the clinical cases in the exanthemata and study the symptoms as they present themselves in many instances.

The writer does not wish to be misunderstood in the matter. He does not claim or believe that all complications in the heart, pleura, lungs, joints, etc., arise from middle-ear disease. Such a position would be absurd. He does believe, however, that a certain number of such complications are of middle-ear origin and the ear may be justly accused of being a possible source of infection until careful examination has proved it free from suspicion.

The etiology of the complications is too well known to need further discussion in so far as extention from the nose and naso-pharynx is concerned. It frequently happens, however, that in scarlet fever both middle ear and mastoid cells become infected long after the acute inflammatory reactions in the nose and naso-pharynx have subsided. In certain other cases, also, it appears that the mastoid becomes infected without symptoms of inflammation in the middle ear. Such being the case, is it not possible to have both middle-ear and mastoid inflammation as a result of primary infection from the blood stream and not secondary to an inflammation in the nose or naso-pharynx? In several of the autopsy records, notes appear which state that the inflammation in the middle ear or mastoid appears to be primary and not secondary to adjacent organs. This is an interesting phase of

the situation but the writer has neither the time nor necessary facts at hand to scientifically prove a theory of primary infection.

The onset of aural complications is fairly constant in measles. It usually develops with the active inflammation in the nose and throat, and patients who escape during this period usually remain free from the complications throughout the disease. On the other hand, it may be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of the acute symptoms to the last day of convalescence. No special rule may be given as to the onset of the complications of diphtheria. On the whole, they occur late rather than early in this disease, but like scarlet fever they may arise at any time during the illness of the patient.

In mild cases, symptoms of aural complications in the exanthemata are much the same as from other common causes. They often appear without apparent discomfort to the patient and usually do not complicate the original disease to any extent. In severe cases, where great prostration is present or considerable elevation of temperature develops, the importance of an aural complication increases ten fold. Judging from my own experience it is not the symptoms present which confuse our diagnosis, but those which are absent. Those present are quickly and easily recognized; but not infrequently important symptoms which we are taught to believe exist in any typical case of either acute otitis media or mastoiditis are entirely absent. In this respect aural complications of the exanthemata present their peculiarities to the greatest extent.

Age is naturally an important factor. Generally speaking, the younger the patient the greater the susceptibility to aural complication. In severe attacks of measles, adults are nearly as susceptible to middle-ear infection as young children and they are even more liable to mastoiditis, if the middle ear becomes infected. Comparatively few cases of aural complications develop in the clinical cases of diphtheria and I cannot recall having seen one case of mastoiditis in an adult in this disease. Judging from the autopsy records, however, I believe they are not as uncommon in severe cases as we suppose them to be.

Pain as a symptom is most unreliable. There seems to be considerable difference of opinion on this subject among different writers. Some claim pain is constant and distressing and others that it is often absent. My experience is that it is frequently absent and that when it occurs it is by no means as great as that encountered from grip, infectious colds, etc.

Temperature is naturally an important symptom and is doubtless the most constant one. It has a wide range, however, and is not by any means to be entirely depended upon. The onset is usually sudden, not with a gradual rise. The drop is equally sudden after paracentesis or spontaneous rupture, but may be delayed some hours before it actually occurs. That is to say, even after paracentesis or spontaneous rupture, the temperature may occasionally remain elevated for twenty-four hours or even longer before a change takes place. When the temperature begins to fall, however, if free drainage has been established, the drop is sudden and not a gradual remission.

Temperature is by no means always present in serious cases. I have operated upon a number of cases of mastoiditis in which there was a large amount of free pus, where the temperature never exceeded 100 degrees. I recall one case in particular wherein the kidneys showed marked inactivity as well as considerable inflammation in both middle ear and mastoid. The temperature in this case was but 99.8 degrees. In this instance, when the mastoid was opened, the kidneys promptly resumed their function. It is difficult to imagine a general systemic infection of middle-ear origin without some degree of elevated temperature. Such a condition is possible, however, and appears to be one of the peculiarities of the exanthemata.

The appearance of the drum membrane differs somewhat in the contagious diseases from similar conditions from other causes. The color is usually less of a fiery red and is more of a grayish pink. At times, even with considerable bulging, it is a slate blue or lead color. The drum membrane when bulging is often considerably thicker than usual and frequently gives a distinct porky sensation when performing paracentesis. This infiltration doubtless accounts for the difference in color as well as the absence of pain in many cases.

In scarlet fever and measles there is seldom any difficulty in diagnosis. The bulging in either of these diseases is extreme. It is in diphtheria wherein the difficulty arises. Aural inflammation in this particular disease is much less active, and, at times, is exceedingly difficult to recognize. The most difficult diagnoses I have ever been called upon to make occurred in complications of the ear in conjunction with diphtheria. The reason for this will be explained in a measure when we consider the question of discharge.

The most important symptom to be noted in the appearance of the drum membrane is the formation of so-called nipples.

They seldom appear until perforation has taken place and in my experience most often occur in the anterior half of the membrane. The condition is occasionally referred to as a "bleb." This is a mistake, as the latter is an entirely different anatomical structure. A bleb is a blister-like formation due to fluid or air separating two layers of the drum membrane. Blebs are not infrequently seen in measles and often give a decided "pop" on being opened, due to the sudden escape of confined air or gas. A true nipple, or nipple perforation, involves the entire thickness of the drum and is a sort of hernia or area of weakness in that structure giving way to pressure. The principal drainage point from the middle ear may, or may not, be through the nipple. In my experience such perforations nearly always occur at points not favorable to drainage, being too high above the floor of the middle ear. Unless great care is exercised, more or less of the entire nipple will slough away leaving a large and permanent opening. Nipples may be expected in both scarlet fever and measles when the amount of discharge is great. The persistence of a nipple perforation with an over-active discharge is one of the most valuable symptoms I know, upon which to base the necessity of a mastoid operation.

Tenderness over the mastoid process is a most unreliable symptom. Certain writers have laid great stress upon it and appear to have considerable faith in it as a diagnostic guide. The mastoid cavity is frequently filled with pus and granulations without any suspicion of tenderness being present. It is at best a treacherous symptom and is to be viewed with suspicion whenever present, regardless of whether the tender area is over the tip, antrum, or other part of the mastoid process. This is particularly true if ice or cold in any form has been applied long enough to overcome vascular engorgement. If present, in conjunction with other prominent symptoms, it naturally strengthens the diagnosis. It must never be assumed, however, that the absence of this symptom rules out the possibility of mastoiditis. This mistake is often made in the contagious diseases and is a frequent cause for impaired hearing if not the actual loss of life.

The color and character of the discharge varies in the different exanthemata. At the onset they are often much the same, but after a day or so usually assume their special characteristics. The discharge from a scarlet fever ear is usually the creamy white of streptococcus pus. Occasionally the yellowish discharge of staphylococcus is encountered. Pus from pneumococcus bacilli

is usually of a dirty, light-brown color and is tremendous in amount. In measles, the appearance of the pus is usually of a light-brown color lacking the creamy nature of the pus seen in scarlet fever. What germ predominates in the average measles case, I am unable to state. In diphtheria, the aural discharge usually shows K. L. bacilli but never as a pure culture. It is mixed with other germs; no special one predominating. Aural discharges in diphtheria are not as active or as persistent as those in scarlet fever or measles, but share with them the ability to convey infection. Aural discharges from all three diseases possess the ability to convey infection to a marked degree. Thus no patient can be wholly free from the danger of spreading contagion when an aural discharge is present.

The amount and character of the discharge is of extreme importance and the nature of the germ predominating will influence the case to a marked extent. The quantity of the discharge is one of the most important factors in an aural inflammation. While it is but an indication or by-product of an inflammatory process, it is nevertheless the most prominent symptom with which we have to deal. By its presence and activity, we have our best demonstration of the phenomena taking place within the middle ear and upon its physical characteristics we must often make our diagnosis.

First, when fluid is secreted it must have proper drainage. Secondly, the drainage must be sufficiently free, not only to overcome the dangers of absorption, but to prevent excessive pressure upon the membrane. The first condition is very well understood and drainage sufficient to overcome absorption of toxines is an every-day practice. The second condition is apparently not so well understood and is still less properly treated.

Let us forget for the moment the matter of septic absorption from the middle ear or mastoid and its attendant dangers, and consider the matter of free drainage from a purely mechanical standpoint.

The pressure necessary to force even a thin watery secretion through a minute opening in the drum is sufficient to considerably distend that delicate structure. As the amount of the secretion becomes greater and increases in its density, both factors act to increase the distension. Thus the ability of the drum membrane to withstand such a pressure is the only factor of safety afforded the future of the hearing apparatus.

Usually, in young children, the first symptoms of mastoiditis to attract attention is a bulging forward of the auricle due to

edema over the mastoid process. It occurs suddenly, often being present in the morning when there was no suspicion of it the previous evening. The swelling is due to free pus under the periosteum or to a deep cellulitis of all the structures covering the mastoid. As a diagnostic symptom it is much the same as tenderness. It is a positive symptom and calls for operation when present, but its absence means little or nothing. In my opinion, edema over the mastoid usually occurs only when a fistula is established from the mastoid cavity outward to the external tissues. It is Nature's method of relieving acute mastoiditis and its very occurrence in a measure is proof we have failed to diagnose an important element in our case. Edema over the mastoid is analagous to an abscess in any other part of the body which is allowed to burst itself. Good surgery does not allow an abscess cavity to burst itself in other parts of the body. Why should this location differ in any way? Many cases of active mastoiditis show no evidence of edema, hence its absence in a case means comparatively nothing in diagnosis.

The most striking variation between the three diseases is the appearance and characteristics of the pus found in mastoiditis at autopsies in diphtheria. In scarlet fever and measles, it is of the ordinary variety and is usually copious in amount. In diphtheria, it is repeatedly described as green, yellowish green, brownish green, or other colors bordering upon a greenish hue; and as thick, tenacious, gummy, gelatinous or semi-solid, etc. The marked peculiarity of the discharge may largely account for the lack of active symptoms which seem to be peculiar to aural complications of diphtheria.

Before taking up the matter of treatment, we naturally come to the question of prevention of aural complications in the contagious diseases. As the infection reaches the middle ear by way of the Eustachian tube, and the nose and naso-pharynx are the seat of active inflammations, they would seem to be the structures to which preventive measures would be aimed. This is a natural conclusion to reach and would be an entirely logical one had not experience and practice proved it to be otherwise. Some years ago in the Boston City Hospital it was a routine practice to irrigate the nose and naso-pharynx one or more times daily with an alkaline solution in all contagious cases wherein there was an over-active secretion in the nose or naso-pharynx. The result was to greatly increase the proportion of aural complications. Such a procedure is never practiced at the present time

and the number of cases of middle-ear inflammation and mastoiditis is materially decreased. Irrigation of the nose and nasopharynx is, in the opinion of the physicians and aurists of this institution, absolutely contra-indicated under any circumstances whatsoever. This includes all sprays and douches as well as actual irrigation.

There is but one true method of preventing aural complications, i. e., through removal of adenoid tissue before the patient contracts a contagious disease. The proof of this statement is quite apparent in the Boston City Hospital by the marked decrease in the aural complications over a few years ago when the removal of adenoids was not as common a procedure as it is at the present time. Other than removing adenoid tissue prior to the attack of the contagious disease, there is no method of prevention of aural complications which accomplishes a result and offsets the risk it involves of developing in the ear precisely what it was intended to prevent.

In the exanthemata, aural complications are usually well established before attention is called to them. Thus, the treatment is almost entirely surgical. In general, it is the same as in the ordinary aural practice with one or two exceptions. The important difference is the low vitality of the patient and the increased virulence of the infection. We may rely less upon nature in such cases than at any other time. Middle-ear inflammation which might be aborted under other circumstances fails to respond to the usual mode of treatment in the contagious diseases. The ordinary methods of treatment also are not available. If the drum membrane is found to be red and inflamed, prompt paracentesis is indicated. Hot irrigations seldom accomplish the desired results. Inflation of the middle ear and post-nasal applications not only avail nothing, but decidedly increase the inflammatory process. The inhalation of hot steam is a safe procedure but usually fails to give relief. Free paracentesis is the only accepted method of procedure and should not be delayed.

The writer is a firm believer in hot irrigations as long as redness and bulging are present after free drainage is established. As the discharge becomes less in amount, it is often better to discontinue the irrigation and substitute warm peroxid. This treatment is especially beneficial if the discharge becomes very thick in its density. Aurists who advise wicking the canal or placing plugs of cotton in that position should take great care to instruct the nurse or mother not to force them tightly into

the canal. Even a moderately tight packing, of any material, increases the tension of the fluid within the drum and decidedly interferes with the free flow of the discharge. This is a common fault in the treatment of aural inflammations and one which is more harmful than is commonly supposed.

If the discharge suddenly ceases when it has been very profuse suspicion should be directed to the mastoid at once. This is not an unusual occurrence and is a dangerous symptom. In such a case the drum is often distended, but free paracentesis fails to establish a free flow of the discharge. This symptom signifies that the active seat of the infection is in the mastoid cells and not the middle ear. A swelling of the mucosa interferes with the free passage from the mastoid to the middle ear. Sufficient pus finds its way from the mastoid to slowly fill the middle ear and cause distension, but the flow is not sufficient in amount to keep the opening in the drum patulous. Hence paracentesis, no matter how free or how often, fails to establish the necessary drainage for any length of time. In such a case the mastoid frequently presents none of the typical symptoms of that disease, but should its cortex be opened it would be found to be in an active state of inflammation and filled with free pus. This is a type of case which eventually bursts itself through the cortex. When several paracenteses have failed to overcome an aural complication and the drum membrane shows signs of overdistension from any cause, the mastoid operation is seriously to be considered regardless of whether temperature, tenderness, edema, swelling of the posterior walls of the canal, etc., are present or absent. When typical symptoms of mastoidities are present, the mastoid operation should promptly be performed. Ice bags or other methods of applying cold are contra-indicated if there is edema over the mastoid process, marked indications of serious complications in the heart, joints, pleurae, kidneys, etc., or if the temperature remains elevated to a dangerous degree in spite of free paracentesis.

Nipple perforations require prompt and vigorous treatment from the first. The ordinary incision frequently fails to relieve the condition regardless of the number of attempts in that direction.

The writer has occasionally had excellent results from two incisions at right angles to each other with the apex of the nipple as the center of the incisions. This double incision is much more apt to remain open than a single one. I am more and

more inclined, however, to open the mastoid whenever a large nipple perforation persists for any length of time, together with an over-active discharge which does not respond to less radical treatment. This is by all means the safest procedure and one which produces the most excellent results. The tension on the drum membrane quickly subsides, the middle ear becomes dry in a short period of time, and the hearing apparatus is saved from a very possible impaired function.

Whenever the middle ear is suspected of being the primary source of infection in complications of a serious nature and free paracentesis has failed to control the aural inflammation, the mastoid should be opened at once. This is particularly true if a temporary relief is obtained from paracentesis.

In this respect, I believe, we are justified in opening the mastoid with a more meager collection of symptoms at hand than in any other situation with which we have to deal.

Up to the present time we have had but two methods of treating aural complications—hot irrigations and paracentesis of the drum membrane. Both methods have often proved inadequate and have resulted in thousands of cases of impaired hearing, if not actual loss of life. So far as I know, there is but one other method at our disposal. The mastoid operation in the hands of a skillful aurist is comparatively a simple matter and one which will speedily and safely overcome most aural complications if performed within a reasonable length of time. As soon as drainage has been established through the mastoid wound, the delicate hearing apparatus of the middle ear is largely relieved of the strain in every way and soon returns to its normal condition. All possibility of septic absorption from both middle ear and mastoid is removed and this danger to the patient's life, or well-being, is also eliminated. If one is to err at all in the matter of performing a doubtful mastoid operation, by all means let it be on the side of safety. To open a normal mastoid is no great error but to fail to open one which is diseased may easily result in the loss of the patient's life.

The time is not far distant when draining an abscess cavity through a pin-hole perforation, in an important structure, will be considered poor aural surgery.

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THE ETIOLOGY, DIAGNOSIS AND TREATMENT OF THE AURAL COMPLICATIONS OF THE EXANTHEMATA.*

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A statement found in practically all of the text-books on otology and upon which peculiar emphasis is laid, is the rapidity with which tissue destruction and profound changes in the hearing faculty occurs in certain of the exanthemata. We read also that a fairly large percentage of the chronic suppurative conditions in the ear may be traced to some antecedent infectious disease as their starting point.

By reason of this teaching which has been handed down from generation to generation many otologists have a feeling of apprehension when called upon to treat the ear in these diseases. That this is justified is in a measure true. The reason for it is that in private practice the attending physician is usually too willing to trust to nature to overcome the aural complications and the otologist does not see the case until the process is well established or serious extension has occurred. In other instances, patients are not seen until a later period when they appear at the office or clinic for relief of a chronic running ear.

As a matter of fact comparatively few of these cases are seen by the aural surgeon in the acute stage except in contagious hospitals. Even in these institutions a spirit of diffidence or carelessness is apt to appear at times and a routine course of cleaning the ears more or less thoroughly at irregular intervals is pursued by some one with little or no training or knowledge in this particular line of work. If these cases were properly treated from the beginning there would be less serious complications and fewer chronic suppurative conditions would result. However, it is admitted that a certain proportion will result unfavorably no matter how skillfully the treatment is administered.

In considering the etiology of the aural complications arising during the course of the exanthemata it may be broadly stated that their number and severity will depend upon the class of patients and the character of the epidemic. Other factors that must be taken in the light of predisposing and contributing causes are the age, climate and season, the physical condition of the patient, the

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condition of the upper respiratory tract, the specific infective disease, cross infections and systemic complications.

Unfortunately we are still in the dark as to the exact causative agent of the various eruptive diseases. The bacteriological findings will vary according to the time in which cultures are made. Late cultures will usually show a mixed infection. In a considerable number of scarlet-fever patients at the Durand Hospital for Infectious Diseases, Chicago, early bacteriological examinations of secretions from the ear, nose and throat showed almost invariably a hemolytic streptococcus with occasionally the streptococcus viridans.

Age: Inasmuch as the exanthemata occur chiefly in childhood we naturally find the greatest number of aural complications occuring up to the age of 12. On account of the anatomical characteristics of the Eustachian tube, infants and very young children are more apt to be affected. The infection also seems to pursue a more chronic course. In adults the percentage of suppurative involvement is very small. We find, however, that many will show evidences of a catarrhal otitis to a greater or lesser degree.

Climate and season: In the colder months there is more liability to severer infection of the upper respiratory tract with a consequent greater possibility for aural extension.

Physical condition of the patient: In hospital practice most of the cases come from the poorer and over-crowded districts. As a result of their environment these patients show the effects of malnutrition and other evils of their unhygienic mode of living even before the contagious diseases appear. We have usually a severer type of disease with a larger number of aural infections in these patients, on account of their lowered physical resistance. Many of them do not enter the hospital until relatively late in the disease, and they show the effects of neglect of the primary condition as well as the complications.

Conditions of the upper respiratory tract: This is practically the most important factor in the causation and the chronicity of the infection of the ear. In the majority of cases, especially of scarlet fever, the initial complaint is that of sore throat. We may have a coincident involvement of the nose and naso-pharynx, or what is more common, an extension of the infection to these parts after a short time. Of the children we have treated at the Durand Hospital, it is rare to find one who has had any attention given to the tonsils and adenoids. As a result we find usually an infection of the post-nasal tissues accompanied by what may be desig-

nated for lack of a finer diagnosis, as a purulent rhinitis. Extension readily takes place to the middle ear. In all cases in children where the discharge from the ear has continued, we have had a persistent post-nasal infection. If provision could be made for the removal of the adenoids before the patient is ready to be discharged, I am confident that we would meet with fewer cases of chronic suppuration which could be attributed to the exanthemata. From the same standpoint another phase to be considered is that of reinfection. Cases which have been discharged as cured, under the influence of subsequent nasal or post-nasal infections, may easily acquire a reinfection of the ears.

The specific exanthem: According to different observers the percentage of aural infections varies within relatively wide limits. One estimate given is that at least one-third of the cases of scarlet fever are affected. Acute suppurative otitis media is regarded as the most common complication of scarlet fever. I have gone over the records of some 247 cases of scarlet fever treated at the Durand Hospital during the past year, and find that thirty-five or a little over 14 per cent had suppurative otitis media. In eighteen cases the condition was bilateral, and in seventeen unilateral. One-third of the total number had discharging ears on admission. Dr. E. K. Armstrong, of the Contagious Department of Cook County Hospital, kindly reviewed the records of 100 patients, and found a total of 23 per cent with suppurative otitis. This illustrates the variability of the statistics in this disease. A condition not emphasized enough is the catarrhal otitis media in scarlet fever. An almost equal number of patients were found to have some aural complaint such as fulness, tinnitus, pain or aching or impairment of hearing. The membranae tympani were congested to varying degrees, or were lusterless and retracted. In several there was an exudate in the cavum tympani, which upon evacuation was sero-sanguinolent and persisted only for a short time. Mastoiditis is not regarded as common in scarlet fever. There is not much doubt, however, that a large number of the cases which pursue a chronic course would be found, if operated upon, to have involvement of the bony structure.

In measles, the ordinary complications are of the suppurative and catarrhal types. The aural involvement usually occurs early in the disease. The frequency of suppurative conditions may be as high as 25 per cent of the cases, and an exudate, either serous or purulent is thought to be present in at least 75 per cent. Mastoiditis is much more common than in scarlet fever.

So much for the clinical side. When we come to review the post-mortem findings in fatal cases of measles and scarlet fever we find that in the former, evidence of purulent inflammation is found in the middle ear in 90 per cent of the cases. In scarlet fever the proportion showing changes in the middle ear and mastoid cells is fully as large. This has led Borden to offer the suggestion that we have in the middle ear a focal point for the dissemination of infection. This is exclusive of cases of lateral sinus involvement. I believe this to be true with limitations. However, in young children dying of other causes than infectious diseases, it is quite common to find pus in the middle-ear cavity. We know, too, that even with apparently normal ears we may have serious complications so that it is a matter of extreme difficulty to decide how much one particular part is responsible for the systematic involvement.

Cross infections: In patients where two or more of the infectious diseases are present, or follow, the percentage of aural involvement rises rapidly. It is estimated that over 50 per cent of these patients will have ear complications. When diphtheria and scarlet are associated, suppurative otitis is very common.

Systemic complications: When nephritis is a complication it has seemed to me that the otitis runs a longer course and does not yield as readily to treatment as in other cases until the irritation of the kidneys subsides. Suppuration of other parts as from a cervical adenitis or arthritis also seems to delay healing, probably on account of its exhaustive effect upon the general constitution. As was stated above, the frequency and severity of the ear complications will depend, to a great extent, upon the character of the epidemic. It is in the septic or malignant types of scarlet fever and measles that we find the most frequent involvement of the ears with serious complications and permanent structural damage. In ordinary cases the severity of the infection will not differ from that which we find produced by other causes. We have pain, temperature, tympanic congestion, distension of the drum membrane and improvement of hearing. The distension will be relieved either by spontaneous perforation or incision and the discharge, usually sero-sanguinolent, will become purulent within thirty-six hours. In other instances the discharge may be purulent from the beginning. Usually there will be a subsidence of symptoms after drainage is established but severe otalgia may persist for a number of days.

When the progress of the case is satisfactory the discharge will last from one to three weeks. The time at which the aural symp-

toms appear is subject to many variations. In general it will be while the infective condition in the throat is at its height. The average time is from the third to the sixth day. In severely septic cases ear symptoms may make their appearance within the first twenty-four hours. In these early cases there is usually a marked involvement of the canal, which may be completely closed by swelling. These cases may show an almost co-incident involvement of the mastoid process or an extension to this part within a short time. In this type we have the rapid tissue destruction. In other cases the middle-ear inflammation may not show itself until the patient is convalescent. I have seen it occur as late as the sixth week.

The changes produced by the inflammatory process may also vary. We see cases that have been comparatively mild clinically leave permanent defects upon the hearing power. Others recover with apparently little or no ill results. In severe forms the tympanic membrane and ossicles may disappear. Necrosis of the mastoid portion and the labyrinth wall takes place. We may have spontaneous hemorrhage from the sinus or jugular bulb, or sinus thrombosis may occur. Brain abscess, meningitis or labyrinthitis may result from the extension of the middle-ear process. Fortunately, these more serious complications are not common. In a certain number of patients we have evidence of profound deafness in the absence of any great amount of destruction or other evidence of labyrinth or meningeal involvement. The only explanation for this condition may be found in attributing it to that of a toxic neuritis involving the auditory nerve.

Diagnosis: There can be no hard and fast rules established. In most cases we arrive at our conclusions in regard to the aural condition in the same manner as in the ear infections from other causes. In very young children the diagnosis is at times attended with considerable difficulty. Either an apathetic condition or fretfulness of varying degree may give one an indication of aural involvement. The examination of the drum membrane will, in most instances, reveal the trouble although it is a well-known fact that an otitis may be present without a definite picture of inflammation of the tympanic membrane or of exudation in the cavum tympani. The holding of the hand to the ear is not always a reliable sign, and in most cases is not present. Neither is the enlargement of the subauricular gland to be depended upon, as a cervical adenitis is usually present from the accompanying throat infection. Pain in the ear is, of course, a warning sign, but in certain instances

this may be referred from a cervical adenitis, the throat infection or a decayed tooth. Infants or young children may give no signs of pain, the discharge from the ear being the first evidence of trouble. The same may apply to older children who are quite ill. The temperature and pulse offer the most reliable evidences of the otitic change. Where sufficient drainage is established, both the pulse-rate and temperature will fall. If drainage becomes obstructed we will have a rise in the temperature. If fever persists in spite of free drainage one must look for complications. When the otitis is complicated with severe infective conditions of other parts, as for instance, cervical adenitis, arthritis, nephritis or pneumonia, our difficulties are magnified. Only the continuous and careful observation of our patient will enable us to come to any conclusion as to the preponderating trouble.

As illustrating the complexity of some of these cases, I recall a patient with mastoiditis and a severe nephritis. After operation, progress was satisfactory for a time, but soon the occurrence of severe pain and headache, localized to the affected side, chills and irregular temperature caused keen anxiety as to the possibility of cranial complications. In this instance an exacerbation of the acute nephritis was found to be at the bottom of the trouble, and as this lessened in intensity, the localizing symptoms abated correspondingly. In severely sick patients where the prognosis is doubtful at best, one hesitates to add operative dangers unless a reasonable expectation of benefiting the patient is present. However, it may be said that when an extension of the suppurative condition occurs, even though the patient has pneumonia or nephritis, or other severe complication, one should not hesitate to operate in spite of the dangers of anesthesia, surgical shock, etc. The chief danger to the patient is in delaying the operative interference too long.

When sinus involvement is suspected it must be remembered that the blood culture loses its diagnostic value to some extent, as there is a definite bacteria in scarlet fever. In an article that will appear shortly in the *Journal of Infectious Diseases,* Drs. Henry and Dick will show that blood-cultures in twenty out of twenty-four scarlet fever patients gave positive results. The differential leucocyte count should be closely watched. Examination of the spinal fluid should be made if evidence of meningeal irritation is present. When mastoid symptoms are indefinite, x-ray pictures should be taken if feasible. A suspicion of mastoid involvement is at times aroused by the appearance of swelling and edema over the

mastoid process which arises from inflammation of the higher cervical glands. In these cases a point of value is that the swelling usually appears below the tip and extends upward. It differs from the usual place of appearance at the upper portion of the mastoid as is found generally in children. Tenderness over the tip may arise from the same cause.

The occurrence of a facial paralysis in the course of a severe otitis accompanying scarlet fever is in most instances an indication for opening the mastoid process and providing for drainage from the middle ear through the antrum. Delay in operating may mean a great deal in-so-far as the permanency or recovery of the paralysis is concerned.

The treatment of the aural complications naturally divides itself into their prevention if possible, and their cure, if they have already made their appearance. Under prevention we must try to alleviate the accompanying nasal and pharyngeal conditions. For the latter flushing of the throat with very mild antiseptic solutions is of value. If douching is employed for the nose it should be carried out very carefully. I do not believe it is to be recommended as a general procedure in children. The employment of bland oils with small amounts of menthol and camphor seems to afford some relief. For the removal of secretions from the nose, swabbing with cotton applicators is of value, as is also the use of a suction pump in young patients. We must also employ measures to minimize as much as possible the constitutional disturbances. Attention to proper elimination, sponging for temperature, the careful regulation of the diet where we have a nephritic irritation, are all necessary if we wish to prevent or influence the local infection favorably.

When the diagnosis of an involvment of the middle ear is made, early treatment will depend upon the conditions present. If the membrane is congested and there is but slight evidence of an exudate present in the cavum tympani, we may try to abort the otitis by the application of heat externally or the installation of a 5 to 10 per cent solution of carbolic acid in glycerin. Irrigations of a hot boric acid solution often seem to be of value, as does also the application of moist dressings. Where bulging is present, incision should be made. If the middle-ear condition is accompanied by a violent inflammatory condition external to the drum, incision should be made early. As a general rule, the earlier a free incision of the drum membrane is made the less likely one is to have large perforations and possible destruction of the drum membrane. Un-

fortunately, in a certain number of cases severely toxic or septic from the beginning, it is impossible to prevent destruction or extension in spite of anything we may do.

After drainage is established, it is important that it be maintained. At times this will necessitate oft-repeated incisions in the membrane. The treatment that I have found to be of the most value consists only in keeping the ear as clean and as dry as possible. For irrigation, we use a solution of boric acid or normal salt. After the ear is cleaned, alcohol in various strengths may be instilled and allowed to remain for a few moments. The ear is then carefully dried and gauze wicks are inserted. These wicks are changed as often as they become saturated. The instructions to the nurses are very firm on this point. In the acute stage of the disease this naturally means a good deal of work, but I feel that in no other way could we get results.

If at the end of several weeks the aural discharge gives no indication of diminishing in amount in spite of the most careful attention the question arises as to what should be the next step. It is true that we may continue the local treatment until the requirements of the Health Department are complied with in regard to the length of time patients with discharging ears are compelled to remain in the hospital. This is not satisfactory from the physical and economic standpoint of the patient, or to others from the possibility of the dissemination of infection from the discharging ear.

In these cases several things may be considered: first, the use of vaccines; second, the correction of conditions in the upper respiratory tract which contribute to a continuation of the infection; third, the mastoid operation to provide for better drainage from the antrum and middle-ear cavity.

As with other forms of treatment, vaccine therapy will have its positive as well as negative results. Too much reliance should not be placed upon it alone. It has seemed to me from its use in other conditions that it has its greatest value when used supplementary to operative procedures. In the absence of clinical signs other than the discharge, we must depend largely upon its amount and character to furnish our indications for operative interference. As a rule, if the discharge is persistent and of a mucoid type, our attention will be drawn to the nose and especially to the naso-pharynx. If the discharge remains profuse and purulent after a number of weeks, we must recognize the fact that the cavum tympani alone cannot be responsible for the excessive amount of pus. The naso-pharynx must also receive the required attention in these

cases, but in general, nothing short of the posterior drainage will suffice to bring about recovery.

A matter upon which there is some difference of opinion is whether it is advisable or wise to remove the adenoids during the convalescent stage of the infectious disease. I think that this depends largely upon the general condition of the patient. If the latter is satisfactory and a clear indication for operation is present, it should be done. Even in those cases which have recovered from the aural infection, the removal of the adenoids is to be recommended.

The treatment for the more serious complications does not differ from that pursued in cases arising independent of infectious diseases. It is needless to say that, wherever possible, treatment should not be neglected from the standpoint of the hearing in any of the cases, catarrhal or suppurative, after recovery from the exanthem.

In conclusion the writer wishes to emphasize what has been stated by others many times. 1. The need for closer co-operation between the attending physician and the aurist in private practice. 2. The necessity of competent aural surgeons in attendance in the Hospital for Contagious Diseases. 3. The isolation of patients in these hospitals at least up to the period of convalescence, lessening the liability to cross-infections. 4. The closest attention to patients, including routine examination of the ears in at least the younger patients and careful observation and treatment when symptoms of aural extension arise.

104 South Michigan Avenue.

Vaccination Against Hay-fever. J. FREEMAN. *Lancet.*, April 25, 1914.

Statistics show that active immunization with pollen vaccine succeeds and that the immunity lasts at least for a year after treatment has been discontinued. Patients with marked constitutional disturbances, asthma or inherited tendency are most benefited.

ED.

TWO CASES OF LOSS OF CALORIC VESTIBULAR REACTION, WITH OPERATIVE FINDINGS.*

DR. EDWARD BRADFORD DENCH, New York City.

The following two cases are rather interesting as showing a sudden ablation in caloric reaction, together with the subsequent labyrinthine findings:

Case 1: A woman, 24 years of age, presented at the New York Eye and Ear Infirmary, with the following symptoms: There had been a discharge from the left ear at intervals, since childhood, with great impairment of hearing. Two weeks before coming to the clinic the patient complained of severe pain in the ear with rumbling noises, which continued up to the time of coming under observation. The patient also complained of dizziness, nausea and vomiting. For the first week the patient vomited about twice daily, but during the last week this had disappeared. The patient was unsteady when standing, and had a tendency to fall backward and to the left side. The patient had headache at the onset of the present trouble. The right ear had been negative, as far as the patient's history was concerned.

An examination of the drum membrane showed that there had been some previous trouble in the right ear. Examination of the left ear showed the canal somewhat narrowed, a slight purulent discharge, the membrana tympani apparently destroyed, and the normal situation of the membrana tympani occupied by a mass of granulation tissues. Slight tenderness anterior to the mastoid tip.

On physical examination the patient appears to be a fairly well developed, adult female, but poorly nourished. There is a slight spontaneous nystagmus on looking to the right. The patient is unsteady and falls backward and to the left side, with eyes closed. Gait unsteady, no ataxic symptoms of arms or legs. Face normal. Knee-jerks active, but not exaggerated. Babinski sign not present. Heart and lungs negative. The caloric test, on admission, showed an active labyrinth on each side. The caloric test on the following day showed no reaction either to the hot or cold water test upon the left side. The

*Read at the Seventeenth International Medical Congress, Otological Section, London, August, 1913.

caloric test on the right side was normal. The patient at this time falls toward the right when walking and jumping, and feels dizzy.

Owing to the fact that the caloric reaction had changed so rapidly in twenty-four hours, that the patient was extremely dizzy, and that the temperature had risen from 99.2 degrees to 100.8 degrees immediately after admission, together with the sudden loss of caloric reaction, the patient was immediately placed upon the operating table. The ordinary radical operation was first performed. A fistula was found in the horizontal semi-circular canal, and the oval window was found open. This apparently explained the labyrinthine symptoms. Consequently, a complete labyrinthine extirpation was performed, according to the Neumann method—that is, by a free exposure of the dura in the middle cranial fossa and a free exposure of the lateral sinus posteriorly. The facial ridge was then lowered to its extreme limit. The bony structures were removed in Trautmann's triangle and the labyrinth entered posteriorly, in the usual way. This drained the labyrinth posteriorly. Anteriorly, the lip of bone separating the oval and round windows was removed, thus draining the first and second turns of the cochlea. Free drainage being established, the ordinary meatal flap was formed, the posterior wound sutured, and the wound dressed in the ordinary way. This patient made an uneventful recovery.

Case 2: Boy, 14 years of age, was admitted to my service at the New York Eye and Ear Infirmary, with a history of discharge from the right ear, of six months' duration. Two months after the appearance of this discharge he came to the clinic, and two months subsequently the discharge ceased. Three days before his admission to the hospital, and six months after the first appearance of the discharge the patient complained of acute pain in the right ear and over the sterno-mastoid muscle. There had been no discharge from the right ear during the present attack. On account of the pain the patient had not been able to sleep for three nights. The patient also complained of headache and dizziness. No symptoms referable to the left ear.

A physical examination showed granulation tissue in the right tympanic cavity, springing from the upper and posterior quadrant. Upon the removal of this granulation tissue, it was evident that the ear had been the seat of a previous chronic suppurative process. There was a cicatricial membrane covering the tympanum and lying at a lower level than the normal drum

membrane. This cicatricial membrane was reddened and somewhat bulging posteriorly. The temperature, at the time of the first examination, was 99.8 degrees.

The patient seemed seriously ill and was therefore admitted to the hospital. The caloric test, made at the time of the patient's admission, showed that both labyrinths were active. The granulation tissue was removed from the right tympanic cavity immediately upon the patient's admission to the hospital. Following this slight operation, the temperature began to rise until in twelve hours it had reached 104 degrees. It remained practically at this level for eight hours.

On examining the patient eight hours after the operation, I found him to be a well-nourished boy, complaining of severe pain in the right side of the neck, and crying out with the almost characteristic meningeal cry. There was no stiffness of the neck, no muscular rigidity in any part of the body, the Babinski and Kernig signs were negative. There was slight nystagmus to the affected side. A careful caloric test showed that the right labyrinth was absolutely dead,—this, in contra-distinction to the examination made twenty-four hours before, which showed an active labyrinth on this side.

This total absence of caloric reaction of the right labyrinth, together with the restlessness of the patient, made me believe the case to be one of meningitis following a labyrinth suppuration, probably excited by the removal of the granulation tissue from the right ear. The high temperature, however, and the absence of all other symptoms of meningitis,—such as the abscence of the Babinski and Kernig signs, the clear sensorium,—aside from the restlessness and crying out of the patient, induced me to make a guarded diagnosis. A spinal puncture at this time showed that the cerebro-spinal fluid was clear and was not evacuated under pressure; the differential count of the fluid was normal, its reaction was slightly alkaline, and the carbohydrate test was normal; the globulin test was also negative. A blood culture made at this time showed after eighteen hours two colonies of streptococci. Three or four hours later the plates showed two or three more colonies.

It was evident, therefore, that we had to deal, in this case, with a general streptococcic infection, and not with a meningitis. A radical operation was performed, and neither the sinus nor dura was exposed at the time of the operation. There was no fistula in any of the semi-circular canals, and the oval and round windows were closed.

Owing to the negative findings, as far as a meningitis of labyrinthine origin was concerned, and remembering the negative findings of the cerebro-spinal fluid—also bearing in mind the high temperature and the streptococcemia—I decided to expose the lateral sinus. The sinus lay fully one-half inch behind the limit of the radical operation, and the bone separating the sinus groove from the radical cavity was hard and dense. Nevertheless, when the sinus groove was opened there was an escape of fetid gas. The sinus was found collapsed and separated from the posterior surface of the petrous pyramid by a space of fully three-eighths of an inch. No free pus was present in this space, although the sinus was covered with an exudate of coagulated fibrin. The sinus was rapidly exposed backward toward the knee and downward to just above the bulb. It was thrombosed throughout this entire area. The internal jugular was then exposed from the level of the omohyoid muscle to the base of the skull. It contained fluid blood. The facial vein was tied off between two ligatures at its junction with the jugular. Just above the level of the facial vein the internal jugular appeared thickened and white in color. In other words, it seemed to be occupied by a clot at this level. The jugular was removed from just above the omohyoid to a point just below the base of the skull. The neck wound was closed in the usual way.

The lateral sinus was opened at a point midway between the knee and the bulb. The sinus wall was very much thickened, but after the lumen of the vessel had been entered and a curette introduced downward, free hemorrhage occurred from below. Hemorrhage was controlled in this direction by packing, and the torcular end of the sinus was curetted, with the result that free hemorrhage occurred. This hemorrhage was controlled by firm packing. The patient's temperature before operation was 105 degrees. It fell within the next twelve hours to 99.2 degrees, and the following day it did not rise above 102.4 degrees. At noon the next day it was 101.2 degrees. It then gradually began to rise, and at midnight on the second day after the operation, it was 102.6 degrees.

At this time the patient began to complain of some pain in the right side. The physical signs showed that he was suffering from either a beginning pneumonia or from pleurisy with effusion. The fact that he had simple crepitation over this side, did not render the diagnosis positive at this stage. Lumbar puncture was made on the third day after the operation. The spinal punc-

ture showed a normal cerebro-spinal fluid. It was clear in appearance, the globulin test was negative, and Fehling's solution reduced. The cellular count was negative except for a few blood cells. A small amount of albumin was present in the cerebrospinal fluid. We assumed, therefore, that the patient was not suffering from meningitis, but that the rise in temperature was entirely due to the chest complication. On the following day, that is, the fifth day after the first operation, the patient's temperature rose at noon to 105.8 degrees. A lumbar puncture showed a clear fluid, but a cultivation of the fluid the following day showed long chains of streptococci. The next day a lumbar puncture showed that albumin was present, the globulins were in excess, and pus, blood and streptococci were present in the cerebrospinal fluid, thus clearly demonstrating that the patient had a meningitis.

The patient was immediately taken to the operating room, and an exenteration of the labyrinth performed, according to the Neumann method, the steps of the operation being the same as those detailed in the previous case. In this case, the superior petrosal sinus crossed the field of operation, but was exceedingly large, and, consequently, it was impossible to open the cerebellar dura at the internal auditory meatus, as advised by Neumann, in these cases. The entire sinus groove was filled with a sloughing sinus wall. The bulb was then thoroughly exposed and curetted, and the patient returned to bed.

The temperature dropped after this operation to 102 degrees, but rose in eight hours to 103 degrees. It then fell to 101.8 degrees and the patient was very comfortable. The pulse was good, and the patient simply complained of pain in the chest. Suddenly severe dyspnea developed, the patient became cyanotic and he died within half an hour of a sudden pulmonary embolus.

These two cases are of interest in that they show a sudden ablation of labyrinthine function—that is, the labyrinth tested at intervals of twenty-four hours showed a complete loss of caloric reaction. This seemed sufficient in each case to warrant the complete labyrinth operation, and the findings at the operation clearly demonstrated the wisdom of such a procedure.

The complete cure in the first case was evidently due to prompt interference. The second case was of unusual interest, in that we had to do with a sinus thrombosis, apparently of long duration, as judged by the operative findings. I do not think there is any question but that the sudden involvement of the labyrinth,

in this case, was secondary to the jugular thrombosis. When we bear in mind the close relation which exists between the aqueductus vestibuli, the aqueductus cochleae and the lateral sinus, it is not difficult to understand why a thrombosis of the lateral sinus would cause a sudden ablation of labyrinthine function. The aqueductus cochleae, as we know, lies just above the jugular bulb. The aqueductus vestibuli occupies the posterior surface of the petrous portion of the temporal bone, just in front of the descending portion of the sigmoid sinus. It seems to me beyond question, therefore, in the second case, that infection of the labyrinth occurred secondarily to involvement of the sinus, and that the sudden ablation of labyrinthine function was dependent upon the sinus thrombosis, and prompt interference in this second case would probably have been followed by results quite as satisfactory as those obtained in the first case, had not pulmonary embolism occurred and caused a fatal termination.

15 East Fifty-third Street.

Packing After Mastoid Operation. E. WEISSMANN, *Rev. hebd. de Laryngol.*, May 9, 1914.

The author compares the routine method of packing with gauze after a mastoid operation, the partial closure with drain and the complete closure and suture after the operation, and shows, from an extensive analysis of these various methods, that the latter should be given the preference. SCHEPPEGRELL.

Involvement of Naso Pharynx. W. S. BRYANT, *Am. Jour. Med. Sci.*, July, 1914, p. 61.

Bryant designates the pharynx as the gateway of almost all human diseases since so many diseases are primarily air-borne. The susceptibility of the naso-pharynx is due to man's assumption of the upright position, the growth and development of the brain and the "retrograde metamorphosis of the nose, face and teeth."

ED.

TERATOMA OF THE PHARYNX.*

DR. CURTIS C. EVES, Philadelphia.

J. K., a male child, 14 months old, apparently well formed and well nourished, was brought to the nose and throat dispensary of the Pensylvania Hospital, September 9, 1911, by his parents, who gave the following history:

When the child was 3 weeks old, following an attack of vomiting, they noticed something protruding from the mouth. The father thinking the child had got a piece of cheese in its mouth tried to pull it out but found it was fast. The child chewed upon the growth for a few minutes, then swallowed with difficulty a few times and the growth disappeared. Their physician assured them that there was nothing in the child's mouth or throat and thought they must have mistaken the child's tongue for a growth. Two weeks later the mother again noticed the growth protruding from the child's mouth, disappearing in a few minutes as before. For two or three successive days, each time after taking nourishment, the growth was noticed after the child gagged or vomited. At irregular intervals from a few hours to two weeks, the child had vomited up the growth, chewed at it a few minutes and then swallowed it out of sight. Between the times the growth was seen the child suffered no discomfort, swallowing and breathing normally. The parents had had the child examined several times by different physicians and each time were assured that it was the child's tongue they had seen.

Examination: From the usual examination of the child's throat, nothing could be seen in the mouth or throat. The child was then held by an experienced attendant, the mouth was kept open with a mouth-gag, gagging was induced by placing the index finger in the child's throat. After two or three attempts, a finger-like growth came up into the throat, doubled upon itself until the free end was liberated. Then it shot forward into the mouth, protruding about one-half inch beyond the lips. It was smooth, pinkish in color, and seemed about the size and shape of the child's middle finger. The attachment was by a pedicle to the right side of the posterior wall just behind the upper end of

*Presented as a candidate's thesis to the twentieth annual meeting of American Laryngological, Rhinological and Otological Society, Atlantic City, June, 1914.

the post-tonsillar pillar. When the gag was removed the child chewed upon the growth for a few minutes and with some difficulty swallowed it completely out of sight, the pedicle being entirely hidden behind the posterior pillar of the right side, the main body of the growth was evidently suspended into the child's esophagus. Two days later the growth was removed. Before giving the child an anesthetic, the mouth was held open for an examination of the throat but no evidence of the growth could be seen. To make sure of getting hold of the growth, the child was made to gag it up. The growth came into the throat and shot forward as it did two days before. The end was caught by a hemostat. When the growth was put on the stretch the pedicle appeared so small that it was excised at the base with curved scissors, without an anesthetic. Very little bleeding occurred. An examination of the throat after forty-eight hours showed only a small white area about the size of a pin head.

The laboratory report furnished by Dr. Longcope is as follows: Total length of specimens 5 cm.; length of the skin covered part 2.9 cm.; width of the skin covered part, widest, 1.1 cm.; width of skin covered part, narrowest, 1.3 mm.; length of the mucous membrane covered part 1.5 cm.; length of the pedicle 6 mm.; width 2 mm.

The skin is perfectly white, soft, and of the texture of the child's neck. It shows downy hair projecting from the follicles at about normal intervals for skin. The mucous membrane is pale pink and very smooth. There is no evidence of glands in gross examination. Definite line of demarcation like that at the child's lip. Pedicle seems to have large vessel in it.

Microscopical examination: Section shows skin to be thin, normal, fully developed and supported by normal subcutaneous connective tissue. The center of the mass consists of subcutaneous fat.

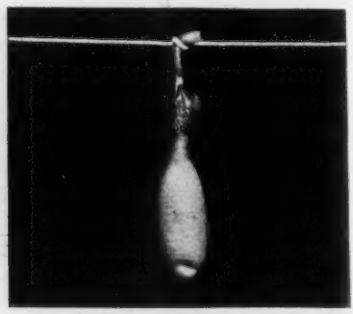
Diagnosis: Teratoma of the pharynx.

Teratomata are mixed tumors of congenital origin formed from the three germinal layers of the embryo. The exact etiology is not definitely determined. They are, as Stengel[1] says, evidently due to some congenital misdevelopment. The most widely accepted explanation as to their etiology is that of Wilms.[2] His theory is that these tumors are derived from the blastomers of the developing ovum, which are supposed to become displaced at a very early stage of development. As these are separated at

a time when differentiation has not yet taken place, it may be assumed that wherever they develop they may, following their inherent developmental power, produce tissue containing products of all three germinal layers.

In pharyngeal teratomata the embryonic layers are represented in varying proportions. Sometimes only two of the three layers are represented. When discovered after early childhood their structures may resemble in parts fibroma, sarcoma or carcinoma, with no other evidence of malignancy. They do not tend to recur when completely removed, neither do they form metastasis.

Teratomata are among the rare tumors found in the pharynx, yet the pharynx is one of the most frequent locations in which growths of this nature are found. This is no doubt because of the development of the pharynx from the close approximation of the germinal layers.



The photograph shows the growth two-thirds its actual size. The depression in the lower end was made by grasping it with the forceps when it was removed.

I have reviewed the literature of twelve cases of teratomatous growths in the region of the pharynx, under the names of teratoma, dermoids and hairy polyps. Out of this number, ten had attachments to the soft palate, one each to the right anterior pillar and right tonsil, and one to the posterior pharyngeal wall. Of the three other cases which have been reported, namely, that of Serapin,[3] Garel,[4] and Aberham,[5] I have been unable to obtain the original reports.

The tumors were generally pedunculated and accordingly gave rise only to symptoms referable to obstruction of the larynx or pharynx, difficult breathing and dysphagia, according to the size and location of the growth.

The ages of discovery ranged from birth to the sixty-sixth year, the greater number being discovered because of dyspnea at birth.

Otto,[6] Clerault,[7] Arnold,[8] Legroux,[9] Mausche,[10] and Kidd[11] reported cases in which it was necessary to remove the growth at birth or within a few days after for the relief of dyspnea. Goscher's[12] case was reported in 1865. The growth was found in the child's mouth after a fit of vomiting, ten days after birth. It caused no symptoms until the sixth month when a tumor the size of a hazelnut, attached by a pedicle, was removed for the relief of difficult breathing during sleep. This case presents especial interest in that Goscher reports a probable recurrence, twelve days after removal, of a similar growth one-fourth the original size. Otto,[6] in discussing Goscher's case gives a very plausible explanation. He says it was probably a portion of the first growth left, or an entirely separate growth attached near, which was not detected at the time the first was removed. He thinks recurrence cannot take place where the growth has been thoroughly taken out.

Fullerton[13] reported a case of apparently two separate tumors, the larger had a pedunculated attachment to the right tonsil, the second, a smaller growth, was attached just below. The microscopical examination of the larger growth showed areas resembling spindle-cell sarcoma and fibroma.

Coffin[14] reports the oldest case, occurring in a woman 66 years old. The base of the growth was dissected out by a unique operation together with the posterior layer of the soft palate. This case showed areas resembling carcinoma. Another case reported by Coffin,[15] giving only symptoms of nasal obstruction on the left side was discovered in a man 35 years old. The growth was attached to the posterior pharyngeal wall.

White[16] removed a dermoid tumor, attached to the soft palate by a broad base, two inches long, the size of one's finger, from a child 3 years old, which only gave symptoms of difficulty in swallowing solid food.

The largest tumor was reported by Kidd[11], a mass eight inches long and six inches in the widest part, protruded from the mouth at birth.

Arnold[8] reported a number of cases in which mixed tumors were found attached in the region of the pharynx in premature dead-born children.

Bosworth[17] says, "It would seem that these cases which have been observed in fetal life were extensive in character and did not belong especially to any region of the fauces, but really involved quite general attachments. The existence of such growths are frequently associated with malformations in other parts of the fetus and that the presence of such a growth was the probable cause of death in the uterus."

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1700 Walnut Street.

Tonsillitis and Appendicitis. L. VERDELET, *Gaz. hebd. des Sci. Med. de Bordeaux*, April 19, 1914.

Case 1: Woman of 36 years; no previous intestinal trouble. Bilateral acute tonsillitis followed by acute abdominal pain intensified at McBurney's point. Vomiting, tympanites, constipation, fever. Recovery under medicinal treatment for appendicitis.

Case 2: Woman of 25 years. Acute sore throat and double tonsillitis. After six days, abdominal pains with symptoms of appendicitis. Improvement and then recurrence; operation. ED.

ORBITAL ABSCESS WITH OPTIC NEURITIS DUE TO ACUTE ETHMOIDITIS IN A CHILD. OPERATION. RECOVERY.*

DR. J. H. GÜNTZER, New York City.

The extreme rarity of optic neuritis due to acute ethmoiditis is sufficient reason for the report of the following case:

Minnie S,, aged 7 years, a normal child and in apparent good health, came to the Manhattan Eye, Ear and Throat Hospital to Dr. Hepburn's Eye Clinic on March 18, 1914. She had had no recent illness until the right eye became swollen two days ago.

There was well-marked orbital cellulitis, the upper and lower lid swollen with tenderness over the inner margin of the orbit, marked exophthalmos with displacement of the eyeball downward and outward, ocular motion limited above and internally, the eye dilated outward. Diplopia: V 20/200. Conjunctival blood vessels injected. Fundus examination showed the veins enlarged, tortuous, the arteries normal. Disc-color=red; margin=blurred. Diagnosis: Optic neuritis.

The patient was turned over to my nose and throat service, and admitted to the ward. On the same afternoon the orbital abscess was incised at the most prominent portion on the nasal side below the brow in order to relieve the ocular pressure. A few drams of pus escaped. The following day the ethmoidal labyrinth was exenterated by the external route. Excepting the sloughing of one of the skin sutures the recovery was uninterrupted. Six weeks after operation the eye-ground has entirely cleared up with V. 20/20.

About two years ago the writer reported and presented to the Section an identical case, in a child, with the same good results following operation.

40 East Forty-first Street.

*Report and presentation of patient at the meeting of the Section on Laryngology and Rhinology of the New York Academy of Medicine, May 27, 1914.

THE USE OF VACCINES IN THE TREATMENT OF CHRONIC DIPHTHERIA CARRIERS.*

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The occasional persistence of the Klebs-Loeffler bacillus in the throats and noses of patients for weeks and sometimes months after their complete clinical recovery, has been a source of annoyance and concern to everyone who has had to do with diphtheria patients. The necessity for complete isolation of such patients as long as the organisms in virulent form are present, self-evident though it is, entails a serious loss of time and inconvenience to the patient.

This is true not only of those carriers who have just recovered from an active diphtheria, but likewise of that other class in whom, though they have never had an active diphtheria, nevertheless either through contact with diphtheria patients or otherwise, the Klebs-Loeffler bacilli are present. The necessity for their isolation is just as great and the presence of the germ is just as persistent, even more so, than in those who have actually suffered from the disease. Needless to say, they are more dangerous than the active case, for the one is carefully guarded against, the other not at all.

To shorten the period of enforced isolation of both classes and to hasten the disappearance of the germs, many procedures have been recommended; antiseptic sprays and gargles for the throat; local applications to the tonsils, especially deep into the crypts; antitoxins; extirpation of the tonsils and adenoids; endotoxins; vaccines and the use in the nose and throat of a spray of a living broth culture of the staphylococcus. In a recent article, Albert[1] has summarized these several methods with sufficient reference to the literature. Brief mention of some of them will be made later.

It is the eradication of the diphtheria bacilli from the nose, throat and ears of both classes of carriers and the resultant stamping out of a diphtheria epidemic in a public institution, an orphan asylum, that forms the basis of the present paper.

After a year or more of sporadic outbreak of the disease, constantly recurring in spite of the most careful isolation of the diphtheria patients, twenty-four cases finally remained in the isolation wards of the Touro Infirmary, where all of the active cases had

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been treated. Doctor Wilson, the visiting pediatrist of the asylum, kindly turned these cases over to me with the request that I should attempt to free them from diphtheria bacilli so as to allow of their return to the asylum.

For the purpose of classification, I have divided the patients into three groups: 1. Those who, having had an active diphtheria infection, had been clinically entirely well for a period of two weeks or longer but in whom live diphtheria bacilli were still found in the cultures. The period during which a positive culture had persisted after complete clinical recovery varied from three months to something over two weeks. There were nine children in this group whom I have called "chronic active carriers."

2. Those in whom, though they had never had an active diphtheria, the cultures showed the presence of the diphtheria bacilli. Since they never had diphtheria and the presence of the bacilli was only discovered in the course of a routine cultural test of all the inmates of the asylum, it is impossible to know just how long they had been carriers. It is fair to assume, however, since the epidemic had persisted for a year or more in spite of the immediate isolation of any true case of diphtheria, that some of them at least had been carriers for a long time and had been the means of infecting many of their less resistant comrades. There were twelve children in this group whom I have called "chronic passive carriers."

3. Those who had an active diphtheria infection but in whom less than two weeks had elapsed from the time of their complete recovery to the beginning of the vaccine treatment. This group comprises three patients whom I have called "active carriers." Since they are not chronic carriers they do not properly belong in this report, but I have included them because I thought it would be interesting to show whether the use of the vaccines had shortened their period of terminal isolation.

Needless to say that this grouping and nomenclature is entirely arbitrary. There is no definite point at which a diphtheria case becomes a chronic carrier. The average time in my experience in which the cultures become negative is about a week after the patient is clinically well, allowed to have solid food and be out of bed. I have taken double this time as the shortest period in which a case might be called a chronic carrier. A glance at the detailed report of cases will show that only Cases 2 and 8 approximate this period, the rest being from four weeks to three months. As for the terms "chronic active carriers" and "chronic passive carriers,"

they are only used to differentiate between groups 1 and 2, and are not suggested as scientifically accurate.

It was a question at the outset just which of the various methods that have been recommended to get rid of the germs in carriers gave the best promise of success. The use of local antiseptic applications and sprays did not appear rational for the double reason that (1) the antiseptic would necessarily have to be too weak to be of much use, and (2) they would not reach into the depths of the tonsil and adenoid crypts, nor into the accessory nasal cavities, the places of refuge, I believe, of the bacilli in these chronic carriers, whence they spread to the superficial mucous membrane and give rise to positive cultures.

The extirpation of the tonsils and adenoids seems to me to be a dangerous procedure in the presence of the living Klebs-Loeffler bacillus and I know of two cases, though they did not occur in my own practice, in which tonsillectomy in carriers was followed by a very severe and serious diphtheria.

The local use of antitoxin in the throat and nose or its use by injection, has given little or no result. It might be well to emphasize the fact, however, that by no means is the use of vaccines or any of the other methods mentioned intended as a substitute for the use of antitoxin in the cure of diphtheria. The great value of that treatment cannot be over-estimated and vaccines are used only to supplement the antitoxin treatment in those cases where the diphtheria bacilli persist after a complete clinical cure has been effected.

Concerning the use of the staphylococcus spray, Catlin, Scott and Day,[2] Lorenz and Ravenal[3] and quite a number of others report success with this method of treatment. Indeed this, though not wholly devoid of danger, seems to be the most promising treatment outside of active immunization of the patients. Those who have tried it report, with few exceptions, no untoward results from its use; but Davis[4] reports a case of acute tonsillitis following the use of the staphylococcus spray. Luckey, quoted by Albert[5] reports a similar case.

A more rational method, it would seem to the writer, is the active immunization of the patients by means of endotoxins or vaccines. If by this means the production of antibodies and other bodies hostile to the growth of the bacilli can be increased in the blood, and if the blood stream can be brought into close enough contact with the living bacilli, their increase would obviously be retarded and eventually they would be destroyed. Theoretically, the efficacy

of this method of treatment depends upon how much effect the hostile blood stream within the tissues would exert upon the living bacilli upon their surface.

Hewlett and Nankivell[6] report a series of twenty-four cases, all of which, except five acute cases, were chronic carriers, treated with endotoxin. They report success in thirteen out of nineteen carriers, and think they noted a shortening of the period of terminal isolation in the acute cases. Even in those chronic cases where the bacilli persisted, they noted a marked decrease in the number of organisms.

The first to use vaccines for the cure of chronic carriers was Petrusshky.[7] He later reports seven cases.[8] In preparing his vaccines the bacilli were killed by means of chloroform vapor instead of heat, and since his vaccines were not standardized either by Wright's or any other method, the dosage in number of dead bacilli is uncertain. In four cases of active diphtheria he reports good results in that the throats were clear of bacilli in one to four weeks. In two chronic carriers one was permanently free of diphtheria bacilli in fifteen months, the other, which showed the bacilli in the sputum as well as in the throat, in about six months. A third case with Klebs-Loeffler bacillus in the sputum, on account of intense reaction and pain at the site of injection, was treated with the vaccine in an ointment rubbed into the skin. The bacilli disappeared in about six weeks. He also recommends the active immunization of children with vaccines, preferably by the inunction method, in order to make them less susceptible to diphtheria. Of several hundred cases thus treated only one has contracted diphtheria.

Hall and Williamson[9] report success in four out of six cases treated with vaccines in increasing doses, from 75 million to 1,000 million. Forbes and Newsholme[10] used vaccine in three cases of membranous diphtheritic rhinitis of long standing, in one of which there was also diphtheritic infection of the ear. Their dosage was from 10 million to 400 million. Although the membrane disappeared from the nose, the bacilli were still present in the cultures but in smaller numbers than before.

In my cases I used first a stock vaccine obtained from one of our manufacturing pharmaceutical houses. The initial dose was in some cases 20 million, in some 40 million, repeated every three or four days in increasing numbers until a maximum dosage of 400 million was reached. The vaccine was administered in every case by subcutaneous injection into the arm. Six doses of the stock

vaccine in all was used on each patient. An interval of twenty days was then allowed to elapse to note what result had been obtained. In the unsuccessful cases treatment was resumed, this time with a vaccine made in the laboratory of the Touro Infirmary from a mixture of the bacilli secured from those remaining positive cases which showed the greatest number of bacilli. The dosage of this, which I call a mixed vaccine in contradistinction to the stock vaccine used above, was begun at 300 million and rapidly increased to 1,400 million, five doses in all of this mixed vaccine being used. The method of preparing both vaccines was similar. After isolation and the growth of the organisms in pure culture, the bacilli were washed from the blood-serum pure culture with normal salt solution, standardized according to Wright's method, killed by a heat of 55° to 60° C. for an hour in a water bath, tested for sterility, diluted to the proper strength and preserved with one-fourth per cent trikresol or one-tenth per cent carbolic acid.

The local reaction at point of injection, though sometimes quite marked, was in no case severe enough to cause any inconvenience to the patient. Several of the patients showed no reaction whatever throughout the entire treatment. A systemic reaction as evidenced by a rise in temperature did not appear. One patient, Case 12, showed a rise of temperature to 100.2° four hours after injection of the initial dose of 40 million, but as there was no local reaction at that time and no increase of temperature or local reaction following the second dose of 80 million, it would seem that the slight rise of temperature after the initial dose was merely a coincidence. There was no apparent relation between the amount of local reaction and the efficacy of the treatment. Some of the favorable cases showed no reaction at all, while some of the most persistent ones gave a marked reaction throughout.

One of the patients, Case 14, developed an acute follicular tonsillitis during the course of the treatment. The cultures made from the throat at this time showed the presence of the staphylococcus in large numbers but no diphtheria bacilli. As the culture made from the throat a few days previous was also free of diphtheria bacilli, this incident throws no light on the question whether, as is maintained by some, a staphylococcus sore throat tends to destroy the diphtheria bacilli. It is on this theory that the treatment by staphylococcus spray is founded.

Of the twenty-four cases, three cases—4, 15, and 20—had cronic suppuratitive otitis media with the bacilli present in the aural discharge. A culture was made from the ear each time it was

made from the throat, and the bacilli disappeared practically simultaneously from both regions. Case 20 had the organisms present in the aural discharge, but not in the nose or throat. Among the chronic passive carriers, five or six had a chronic nasal discharge, though in no case was there a membrane present in the nose except in Case 6 of Group 1, which had a nasal membrane and very little in the pharynx. The cultures from the noses as well as from the throats of these patients were positive. It might be mentioned in passing that in all of the cultural tests of the patients comprising this report swabs were in every instance taken from the nose as well as from the throat. A negative culture throughout this report means that both the nose and throat were negative.

The results obtained from the vaccine treatment although in the early stages of the experiment somewhat disappointing were in the end, I think, sufficiently satisfactory to warrant their further trial in the treatment of chronic carriers. In discussing the result of treatment the three cases comprising Group 3, since they are not chronic carriers, will be omitted at this time, but will be mentioned separately later.

No culture was taken from any of the cases until several days after the third dose of vaccine, 160 million, had been administered, allowing a period of over ten days for the vaccine to have effect. It will be seen from the case-reports that six cases—1, 3, 9, 13, 18 and 22—showed negative cultures at that time, which remained permanently negative. Of these cases which may be called cures, the first three had been chronic active carriers for respectively three, four and seven weeks, and the last three passive carriers for an uncertain time. The objection may naturally be made that these results are not conclusive since the throats might have become clear of bacilli in that time without the use of vaccines. This argument cannot be controverted except that the almost uniform success which later attended the use of larger doses of vaccines and the diminution of the number of organisms in the cases which remained positive, might lead us to believe that they had an influence at least on some of the cases.

At the time of this first trial eight others—Cases 2, 4, 5, 7, 8, 16, 17 and 20—in addition to the six mentioned above gave a negative culture. This led us to expect that the experiment would prove a brilliant success and that the cultures of all would soon be negative. In this hope we were disappointed, for, on the second, third or fourth trial, all of them again showed a positive culture. A glance at the case-reports will show that it was not at all unusual

for two or three negative cultures to be followed by a positive or *vice versa*. This seeming inconsistency was not due to carelessness either in taking the swabs or examining the cultures. The swabs were painstakingly made in every instance, going deeply into the tonsillar crypts or rubbed carefully into the nasal mucous membrane and every slide was thoroughly examined. Thanks are due to Drs. Duval and Langford, the pathologists, and to Dr. Meadows, the pathological interne, for their conscientious assistance in this part of the work. This alternation of positive and negative cultures may have been due to a reinfection of the negative by the positive cases, though the positive ones were isolated as well as possible throughout the course of the treatment. It can be accounted for more probably, however, by the possibility of the few diphtheria bacilli in the culture having been overlooked in spite of the carefulness of the examination; for it was noted in all of the cases shortly after the beginning of the vaccine treatment, that even when the diphtheria bacilli persisted they became so few in number that they could be detected only after a rather careful search of the slide. Previous to the vaccine treatment many of the cases showed the presence of the bacilli in large numbers, sometimes almost in pure culture.

On account of this tendency to recurrence of a positive culture after several negatives it was deemed advisable not to discharge any patient as cured until after he had given at least six negative cultures in succession, nor was the vaccine treatment discontinued in most cases until the occurrence of six consecutive negative cultures.

A further reference to the case-report will show that six more cases—Cases 8, 14, 17, 18, 20 and 21—all of them except the first belonging to Group 2, were cured at the close of the first period of treatment; that is the treatment with the stock vaccines. To most of these, since they had not yet shown six consecutive negative cultures, the mixed vaccines were later given in order to insure the permanency of the cure.

There remained then nine cases in which the diphtheria bacilli still persisted on May 2, twenty days after the finish of the first course of treatment; that is treatment with the stock vaccines, and a maximum dosage of 400 million. This was six weeks after the use of the initial dose of vaccine. On that date the treatment was resumed in these remaining cases with mixed vaccine, beginning with 300 million and repeated every three or four days until a maximum dose of 1,400 million was reached, i. e., five doses of the

mixed vaccines. The final dose was given on May 20. By that time all but five gave permanently negative cultures. These five gave positive cultures on one, or at most, two more trials, after which their cultures were also permanently negative. That they have remained so is evidenced by the fact that up to the time of the present writing, a period over five months, no case of diphtheria has developed in the asylum whither these patients were returned. A period of about two months had elapsed from the beginning of the treatment until all of the patients gave permanently negative cultures.

Of the three active cases comprising Group 3, in one case ten days, and in the other two about one month elapsed between their complete clinical recovery and the last positive culture. Their period of terminal isolation accordingly did not seem to be diminished. The cases, however, are too few to allow any definite conclusion to be drawn.

In drawing conclusions as to the value of the vaccine treatment in the twenty-one chronic carriers, the possibility must not be lost sight of, that many, if not all of them, might have cleared up without the use of vaccines. In view of the fact, however, that practically all of the cases showed a marked diminution in the number of bacilli present shortly after the treatment was begun and that all of them eventually did clear up with the larger doses, the belief would seem to be justified that the vaccines are of some value. At any rate, it has been shown that diphtheria vaccines in large doses can be used without the slightest inconvenience to the patient, and we are justified at least in recommending their further trial whenever the occasion arises.

From our experience it would appear that large doses give better results than small. Whether the autogenous vaccines are more useful than the stock is a matter of opinion. In our experiment we began with a stock vaccine and continued with a mixed vaccine as described above, which although in no sense autogenous was still somewhat different from the stock vaccine.

If we had had a larger number of cases to treat, or if all of them had been chronic carriers for a period of three months or more as some of them were, our conclusions would have been more valuable.

This is a subject of such general interest and so little work has been done along the lines indicated, that I thought it might be worth while to get an expression from a number of men of their opinion as to the existence of chronic carriers and the efficacy of the use

of vaccines in general. I accordingly sent letters to one hundred representative ear, nose and throat specialists throughout the country asking their answers to these questions. Of almost sixty answers received, forty-three believed in the existence of chronic diphtheria carriers, one did not and four were uncertain. Fifteen declined to express an opinion on account of lack of experience. Concerning the efficacy of vaccines, thirty-one believed in their usefulness, twelve were uncertain and sixteen had not sufficient experience to express an opinion.

GROUP 1: Patients who, having had diphtheria had been clinically entirely well for a period of two weeks or longer before the vaccine treatment was commenced. Called "chronic active carriers."

Case 1: G. S., age 6; female; admitted February 20, discharged May 5. February 20, admitted. Active faucial diphtheria. Culture +. February 21, antitoxin, 10,000 units. March 1, clinically well. Since then a carrier. Culture, +. March 1 to 22, culture positive on several trials. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 2, culture —. April 3, stock vaccine, 320 million. April 8, stock vaccine, 400 million. Culture —. April 12, stock vaccine, 400 million. April 14, since then six negative cultures. May 5. Discharged. Culture —.

Case 2: M. S., age 12; female; admitted February 28, discharged June 9. February 28, admitted. Active faucial diphtheria. Culture +. February 28, antitoxin 10,000 units. March 1, antitoxin 10,000 units. March 4, clinically well. Since then a carrier. Culture +. March 6 to 22, culture positive on several trials. March 22, stock vaccine 40 million. Culture +. March 26, stock vaccine 80 million. March 29, stock vaccine 160 million. April 3, stock vaccine 320 million. April 8, stock vaccine 400 million. April 12, stock vaccine 400 million. Culture —. April 14, culture —. April 23, culture +. May 1, culture —. May 2, mixed vaccine 300 million. May 6, mixed vaccine 500 million. Culture +. May 10, mixed vaccine 1,000 million. May 12, culture +. May 15, mixed vaccine 1,000 million. May 16, culture —. May 20, mixed vaccine 1,400 million. May 21, culture +. May 25. Since then six negative cultures. June 9. Discharged. Culture —.

Case 3: F. K., female; age 15; admitted February 13, discharged April 16. February 13, admitted. Active faucial diphtheria. Culture +. February 13, antitoxin 15,000 units. February 14, antitoxin, 10,000 units. February 15, antitoxin 10,000 units. Feb-

ruary 22, clinically well. Since then a carrier. Culture +. February 22 to March 17, culture positive on several trials. March 17, stock vaccine 20 million. Culture +. March 21, stock vaccine 80 million. March 25, stock vaccine 160 million. March 28, culture —. March 29, stock vaccine 320 million. Culture —. April 2, culture —. April 10, culture —. April 12, stock vaccine 400 million. Culture —. April 14, culture —. April 16, discharged. Culture —.

Case 4: C. G.; female; age 10; admitted January 18, discharged June 3. January 18, admitted. Faucial, nasal and aural diphtheria. Culture +. January 25, clinically well. Since then a carrier. January 25 to March 17, culture positive on frequent trials. March 17, stock vaccine 20 million. Culture +. March 21, stock vaccine 80 million. March 25, stock vaccine 160 million. March 28, culture —. March 29, stock vaccine 320 million. March 30, culture —. April 2, culture —. April 10, culture —. April 12, stock vaccine 400 million. April 13, culture +. April 23, culture +. May 1, culture —. May 2, mixed vaccine 300 million. May 6, mixed vaccine 500 million. Culture +. May 8, mixed vaccine 1,000 million. May 12, culture —. May 15, mixed vaccine 1,000 millino. May 16, culture —. May 20, mixed vaccine 1.400 million. May 21, culture —. May 25, since then culture negative on three trials. June 3, discharged. Culture —.

Case 5: B. R.; female; age (?); admitted January 23, discharged June 7. January 23, admitted. Practically clinically well at the time of admission. Culture, +. Has been a carrier since. January 23 to March 17, culture positive on several trials. March 17, stock vaccine 40 million. Culture, +. March 21, stock vaccine, 80 million. March 25, stock vaccine, 160 million. March 28, culture, —. March 29, stock vaccine, 320 million. Culture, —. April 10, culture, —. April 12, stock vaccine, 400 million. Culture, —. April 14, culture, +. April 23, culture, +. May 1, culture, —. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. May 16, culture, +. May 20, mixed vaccine, 1,400 million. May 21, culture, —. May 25, since then culture negative on six trials. June 7, discharged. Culture, —.

Case 6: A. U.; female; age 5; admitted December 17, 1912, discharged June 4, 1913. December 17, pharyngeal and nasal diphtheria. Culture, +. December 25, clinically well. Since then a carrier. Culture, +. December 25 to March 22. culture positive

on repeated trials. March 22, stock vaccine 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 2, culture, +. April 3, stock vaccine, 320 million. April 8, stock vaccine 400 million. April 10, culture —. April 12, stock vaccine, 400 million. April 14, culture, +. April 23, culture, —. May 1, culture, —. May 2, mixed vaccine, 300 million. May 6, mixed vaccine, 500 million. Culture, +. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine 1,000 million. Culture, —. May 20, mixed vaccine 1,400 million. May 25, culture, —. May 29, culture, —. June 4, discharged. Culture, —.

Case 7: A. E.; female; age (?); admitted January 27, discharged June 7. January 27, admitted. Active faucial and nasal diphtheria. Culture, +. January 27, antitoxin 10,000 units. February 3, clinically well. Since then a carrier. Culture, +. February 3 to March 17, culture positive on several trials. March 17, stock vaccine, 20 million. Culture, +. March 21, stock vaccine 80 million. March 25, stock vaccine, 160 million. March 28, stock vaccine, 320 million. Culture, —. March 30, culture, —. April 2, culture, +. April 5, culture, —. April 10, culture, —. April 12 stock vaccine, 400 million. April 14, culture, —. April 23, culture, —. April 26, culture, —. April 28, culture, +. May 2, mixed vaccine, 300 million. Culture, +. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, +. May 20, mixed vaccine, 1,400 million. Culture, —. May 21, since then culture negative on six trials. June 7, discharged. Culture, —.

Case 8: D. H.; female; age (?); admitted February 24, discharged May 17. February 24, admitted. Faucial diphtheria. Culture, +. March 2, clinically well. Since then a carrier. March 2 to March 17, culture positive on several trials. March 17, stock vaccine, 20 million. Culture, +. March 21, stock vaccine, 80 million. March 25, stock vaccine, 160 million. March 29, stock vaccine 320 million. Culture, —. March 30, culture, +. April 3, stock vaccine, 320 million. Culture, —. April 6, culture, +. April 8, stock vaccine, 400 million. April 10, culture, —. April 12 stock vaccine, 400 million. April 14, culture, —. April 25, culture, —. May 1, culture, —. May 2, mixed vaccine, 300 million. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. May 17, discharged. Culture, —.

Case 9: H. P.; female; age 17; admitted January 30, discharged April 17. January 30, admitted. Faucial diphtheria. Culture, +.

January 30, antitoxin 10,000 units. February 2, clinically well. Since then a carrier. February 2 to March 22, culture sometimes positive, sometimes negative on repeated trials. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. Culture, —. April 12, stock vaccine, 400 million. Culture, —. April 14, since then culture negative on five trials. May 5, discharged. Culture, —.

GROUP 2: Patients who never had an active diphtheria, but in whom the diphtheria bacilli were found in the course of a routine cultural test. The length of time they had been carriers is uncertain. Called "chronic passive carriers."

Case 10: P. M.; female; age 7; admitted March 13, discharged May 5. March 13, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. April 10, culture, —. April 12, stock vaccine, 400 million. April 14, since then culture negative on seven trials. May 5, discharged. Culture, —.

Case 11: I. G.; female; age 12; admitted March 18, discharged June 11. March 18, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, +. April 8, stock vaccine, 400 million. April 10, culture, —. April 12, stock vaccine, 400 million. April 14, culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, +. May 6, mixed vaccine, 500 million. Culture, +. May 10, mixed vaccine, 1,000 million. Culture, —. May 15, mixed vaccine, 1,000 million. Culture, +. May 20, mixed vaccine, 1,400 million. Culture, —. May 25, culture, +. May 29, since then culture negative on six trials. June 11, discharged. Culture, —.

Case 12: M. K.; female; age 12; admitted March 18, discharged June 11. March 18, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Temperature reaction to 100.2° four hours after injection. No local reaction at site of injection. March 26, stock vaccine, 80 million. No reaction, local or general. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. Culture, —. April 12, stock vaccine, 400 million. April 14, culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, —.

May 6, mixed vaccine, 500 million. Culture, +. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, +. May 20, mixed vaccine, 1,400 million. Culture, —. May 21, culture, —. May 25, culture, +. May 29, since then culture negative on six trials. June 11, discharged. Culture, —.

Case 13: L. S.; female; age 8; admitted March 13, discharged June 11. March 13, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 2, stock vaccine, 320 million. Culture, +. April 8, stock vaccine, 400 million. April 12, stock vaccine, 400 million. Culture, —. April 14, culture, +. April 23, culture, —. May 2, mixed vaccine, 300 million. Culture, +. May 6, mixed vaccine, 500 million. Culture, +. May 10, mixed vaccine 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, +. May 20, mixed vaccine, 1,400 million. May 21, culture, +. May 25, culture, —. May 29, culture, —. June 2, culture, +. June 4, since then culture negative on six trials. June 11, discharged. Culture, —.

Case 14: N. M.; female; age 11; admitted March 18, discharged May 21. March 18, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, +. April 8, stock vaccine, 400 million. April 12, stock vaccine, 400 million. Culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Developed acute follicular tonsillitis at this time lasting three of four days. Culture, —. May 10, mixed vaccine, 1,000 million. Culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 21, discharged. Culture, —.

Case 15: A. W.; female; age 11; admitted March 18, discharged May 5. March 18, admitted. A carrier, throat and ear. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. Culture, —. April 12, stock vaccine, 400 million. April 14, since then five negative cultures. May 5, discharged. Culture, —.

Case 16: E. G.; female; age 9; admitted March 19, discharged June 11. March 19, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million.

April 3, stock vaccine, 320 million. April 8, stock vaccine, 400 million. Culture, —. April 12, stock vaccine, 400 million. April 14, culture, —. April 23, culture, —. April 26, culture, —. April 28, culture, +. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. Culture —. May 15, mixed vaccine, 1,000 million. Culture, +. May 20, mixed vaccine, 1,400 million. Culture, +. May 25, culture, +. May 29, since then six negative cultures. June 11, discharged. Culture, —.

Case 17: F. B.; female; age 13; admitted March 18, discharged May 27. March 18, admitted. A carrier. Culture, +. March 24, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. April 10, culture, —. April 14, culture, +. April 23, culture, —. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. Culture, —. May 15, stock vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 21, discharged. Culture, —.

Case 18: J. S.; female; age (?); admitted March 18, discharged May 27. March 18, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, +. April 8, stock vaccine, 400 million. Culture, —. April 12, stock vaccine, 400 million. Culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 25, culture, — May 27, discharged. Culture, —.

Case 19: L. E.; female; age 9; admitted March 13, discharged May 5. March 13, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. April 12, stock vaccine, 400 million. Culture, —. April 23, since then five negative cultures. May 5, discharged. Culture, —.

Case 20: J. G.; male; age 11; admitted March 13, discharged May 27. March 13, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine,

80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. Culture, —. April 8, stock vaccine, 400 million. April 12, stock vaccine, 400 million. April 14, culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. Culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 21, culture, —. May 27, discharged. Culture. —.

Case 21: E. S.; female; age 12; admitted March 18, discharged May 30. March 18, admitted. A carrier. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. April 8, stock vaccine, 400 million. Culture, +. April 12, stock vaccine, 400 million. Culture, +. April 23, culture, +. May 2, mixed vaccine, 300 million. Culture, +. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. Culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 25. Culture, —. May 29. Culture, —. May 30, discharged. Culture, —.

GROUP 3: Patients in whom less than two weeks had elapsed between their complete clinical recovery and the beginning of the vaccine treatment. Called "active carriers."

Case 22: J. B.; male; age 8; admitted March 19, discharged May 22. March 19, admitted. Active faucial diphtheria. Culture, +. March 19, antitoxin, 10,000 units. March 25, clinically well. Culture, +. March 29. stock vaccine, 40 million. April 3, stock vaccine, 80 million. Culture, +. April 8, stock vaccine 160 million. April 10, culture, +. April 12, stock vaccine, 320 million. April 14. culture, —. April 23, culture, +. May 2 mixed vaccine, 300 million. Culture, —. May 6, mixed vaccine, 500 million. May 8, culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture, —. May 22, since then four negative cultures. May 28 discharged. Culture —.

Case 23: H. S.; male; age 14; admitted March 3, discharged May 5. March 3, admitted. Active faucial diphtheria. Culture, +. March 3, antitoxin, 10,000 units. March 4, antitoxin, 10,000 units. March 12, clinically well. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, 320 million.

Culture, —. April 8, stock vaccine, 400 million. April 10, culture, —. April 12, stock vaccine, 400 million. April 14, since then six negative cultures. May 5, discharged. Culture, —.

Case 24: B. R.; female; age 13; admitted March 13, discharged May 23. March 13, admitted. Practically clinically well. Culture, +. March 22, stock vaccine, 40 million. Culture, +. March 26, stock vaccine, 80 million. March 29, stock vaccine, 160 million. April 3, stock vaccine, 320 million. April 8, stock vaccine, 400 million. April 10, culture, —. April 12, stock vaccine, 400 million. April 14. culture, +. April 23, culture, —. May 1, culture, —. May 2, mixed vaccine, 300 million. May 6, mixed vaccine, 500 million. Culture, —. May 10, mixed vaccine, 1,000 million. May 12, culture, —. May 15, mixed vaccine, 1,000 million. Culture, —. May 20, mixed vaccine, 1,400 million. Culture. —. May 23, discharged. Culture, —.

Conclusions: 1. Chronic diphtheria carriers do exist. 2. The use of vaccines does have an influence in destroying the bacilli of chronic carriers. 3. The number of bacilli is markedly diminished by the use of vaccines even where they do not entirely disappear. 4. Diphtheria vaccines, in spite of the toxins they contain, can be used in comparatively large doses, without causing a rise of temperature or other evidence of a general reaction. 5. There seems to be no relation between the amount of local reaction and the efficacy of the treatment. 6. Large doses of the vaccines seem more efficacious than small doses. 7. It is necessary to get a number of consecutive negative cultures before a cure can be said to be effected.

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602 Perrin Building.

AN APPARATUS DESIGNED FOR THE PURPOSE OF GIVING AN APPROXIMATELY ACCURATE QUANTITATIVE HEARING TEST.*

DR. JESSE WRIGHT DOWNEY, JR., Baltimore.

In presenting an apparatus that I have designed and found of use in testing hearing it is obvious that the basic principle upon which its value depends is one universally accepted. I, therefore, shall make but small claim to originality, but shall rather endeavor to remind you of the accuracy of the quantitative tuning-fork tests from a practical standpoint.

I especially refer you to the valuable contribution concerning this subject: "Remarks On Tuning-fork Tests, Particularly in View of the Rate of Decrement," presented by Dr. Dundas Grant before the Ninth International Otological Congress (Boston, 1912), and published in the Transaction of that Congress.

The apparatus here presented, (Figure 1), consists of a C^2 tuning-fork (Hartman 512 d. v. s.)[1] tightly clamped and inclosed within a box from which leads a Y-shaped tube attached to a sensitive diaphragm (Bowles' stethoscope) set in front of the fork. The tuning-fork is set to vibrating by means of a rubber-tipped weight allowed to fall by gravity through a tube of known height (Figures 2 and 3). By having the tube placed slightly off the perpendicular an oblique rebound of the weight is obtained, which prevents the occurrence of a second impact of the weight against the fork. The note is clear, free from overtones and can be perceived by the normal ear for thirty seconds.[2]

To use the instrument, one ear-piece of the Y-shaped tube is placed in the patient's ear, the other in the ear of the physician and the fork is set in vibration by allowing the weight to drop

*Presented as candidate's thesis to the twentieth annual meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, June, 1914.

1. The tuning fork used in this apparatus has been tested in the Physical Laboratory of the Johns Hopkins University by Prof. J. H. Anderson, and the percentage value computed according to the formulae advised by Dr. Dundas Grant. See Appendix.

2. An average. After hundreds of tests I have found a variation of from five to fifteen seconds in the duration of the normal perception of this note. It is the difference in time of perception between the defective and the normal ear which is of importance, and not the duration of the note; therefore, so long as this difference remains the same the duration of the sound is unimportant.

through the tube, both the patient and the doctor thus receiving the sound under exactly similar conditions. The patient is instructed to say when the note is no longer heard and the duration of hearing is recorded on a stop-watch that has been started the moment the weight struck the fork. The duration of the note as further perceived by the examiner is also marked, and the result of the test is written thus:—e. g. —5"(25/30), the numeral denoting the *difference* in the duration of perception and being the important index; the fraction indicating the duration itself, defective and normal, and being of but secondary interest. The test is repeated several times and if the *difference* between the duration of the patient's and the physician's perceptions of the sound remains the same, the test may be considered accurate and the percentage of actual hearing-power for this particular note, under surrounding conditions, as compared to the examiner whose hearing is assumed to be normal, may be read off the table of percentage values, prepared for this apparatus.[3] (See Table.)

TABLE I.

RATIO OF AUDITORY PERCEPTION EXPRESSED AS PERCENTAGES.

Difference in time of hearing.	C^2 Fork. 512d.v.s.	Difference in time of hearing.	C^2 Fork. 512d.v.s.
0	100%	—8 "	3.6%
—1 second	66%	—9 "	2.3%
—2 seconds	43%	—10 "	1.5%
—3 "	28%	—12 "	0.6%
—4 "	19.5%	—15 "	0.1%
—5 "	12.5%	—20 "	0.002%
—6 "	8.2%	—25 "	0.0003%
—7 "	5.4%	—30 "	0.000000

After four years' use of this apparatus in a noisy clinic I feel convinced that it is superior to the other methods of quantitatively measuring hearing, (voice, watch, Politzer and similar acoumeters). And although it by no means eliminates the shortcomings of these methods, and is open to the same criticism that it tests but a restricted portion of the basilar membrane, it furnishes one index of more constant and uniform value for recording and comparing, from time to time, variations in audition,

3. I test a patient under examination, noting: 5"(15/20)(20/25)(25/30). The duration of hearing has markedly varied but the difference in the duration of perception has remained the same. By reference to the table, I find that the patient's hearing-power is reduced to 12.5 per cent as compared to my hearing-power for the same sound, heard under exactly similar conditions. Under treatment, the patient improves and my tests show,—3"(20/23)(17/20)(27/30.) The patient's hearing-power for this same note wil have advanced to 28 per cent as compared to my hearing which I assume to be normal.

and thereby the progress or retrogression of a case under treatment. The ratio, or difference, between defective and normal perception of the duration of the sound is almost always most accurately preserved, and this as we know is *the* important factor in estimating the percentage of actual hearing-power for the note of a tuning-fork.

If the decrement of vibration of a tuning-fork took place in arithmetical progression, so that the duration of perception corresponded to the actual hearing-power, we would have a very simple way of calculating the amount of hearing for a certain tone. As a matter of fact, the decrement takes place by a diminution of the amplitude of vibration in geometrical progression which is governed by the amplitude of vibration of the par-

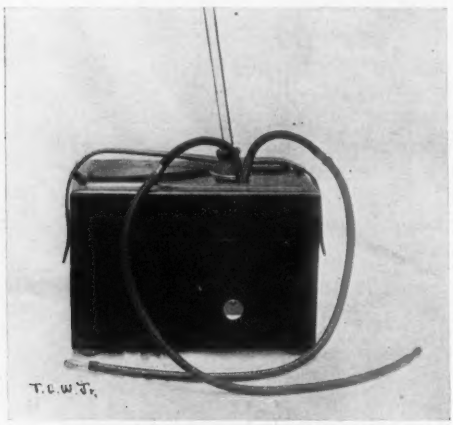

Figure 1. Photograph of "tone-meter" closed.

the beginning but slower toward the end. Accordingly the actual power of hearing is much smaller than the duration of hearing.

Otology is certainly indebted to Dr. Dundas Grant for his scientific and accurate method of ascertaining the percentage values of the auditory perception of the notes of tuning-forks. It has seemed to me that his investigations can be made of great practical value in establishing a system of quantitatively testing hearing which, if not uniform, may at least be generally intelligible. May I, therefore, briefly quote from the essay to which I have previously referred?

The ratio of acuteness of hearing, says Dr. Grant, does not depend in any way on the initial loudness but on the difference of time of hearing and on the constant K relating to that fork.[4]

"The experimental determination of the constant *K* for any given tuning-fork can be carried out at any physical laboratory and the calculation can be made by anyone accustomed to use logarithms in the course of his business." (Grant, page 455, Transactions Ninth International Otological Congress.)

The constant *K* has been determined for the fork used in my "tone-meter" and from it the table of percentage values constructed; therefore, should I read in an article by Dr. Grant, that a patient's hearing for the 512 d. v. s. showed a difference

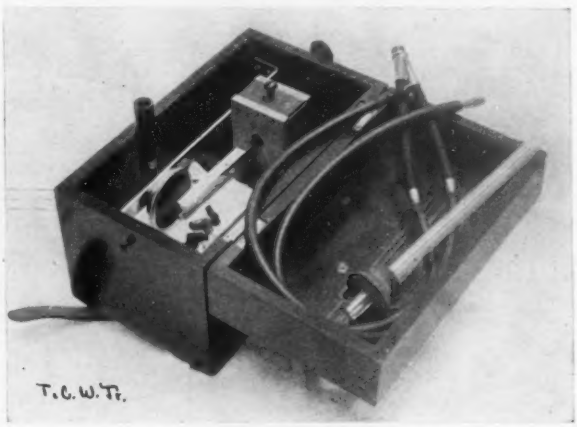

Figure 2. Photograph of "tone-meter" open.

of —5 seconds or 5.9 per cent actual hearing-power for this note, by a reference to my own table I should find that this corresponded to —7 seconds with my fork.[5] Certainly the use of the tuning-forks in the way suggested by Dr. Grant is more than "a step toward accuracy," as he modestly terms it, and should be universally understood.

The advantages of the "tone-meter" over the use of the tuning-fork in the ordinary way have seemed as follows: 1. The constant *K*, necessary in the determination of the percentage of

4. The "constant K" is the term used to denote "the time rate of the decrease of the logarithm of the loudness."

5. Grant's tuning-fork 512 d. v. s., constant K,=0.6294. Tuning-fork (512 d. v. s.) used in my "tone-meter," constant K=0.415.

hearing-power, once being ascertained, is rendered constant for the fork in use. 2. The patient and the physician receive the sound under precisely similar conditions. 3. The ratio between the patient's and the physician's perception-duration of the note is more exactly preserved; and this is not only of practical value as a means of recording the result, but the important and essential factor on which is based the determination of the actual hearing-power as compared to that of the examiner's for the same sound. 4. The method of setting the fork into vibration is practically uniform.

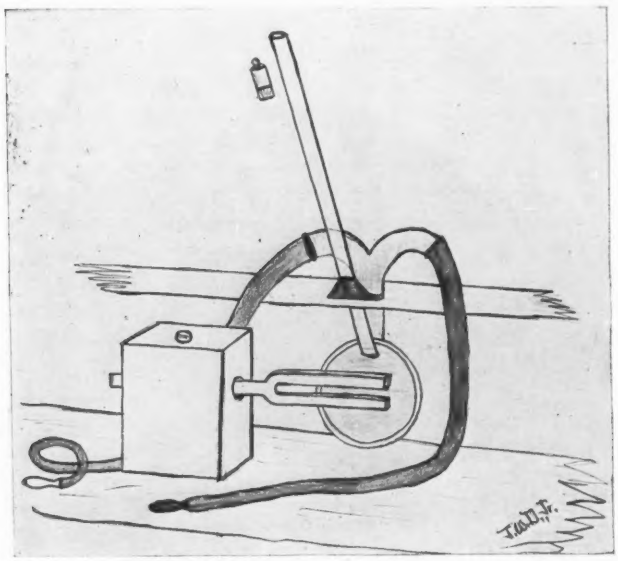

Figure 3.

Its advantages over the other method of quantitatively measuring audition: 1. It gives a more constant and uniform standard of value under adverse external conditions: i. e., noisy surroundings. 2. It is less liable to be misinterpreted by the patient whose answers are more likely to be definite and to the point.

Finally, let me emphasize the limitation of this apparatus. I have found it superior to the voice, the watch and the Politzer acoumeter. It is a plea for the use of the tuning-fork quantitatively as well as qualitatively, that is all.

I have under construction an apparatus to use a C (128 d. v. s.) and a C^1 (256 d. v. s.); the C^2 fork used in this apparatus being too high in pitch to judge accurately the hearing for speech.

There is also the question of the conductivity of the tubes, and what part of the perception of the sound may be due to direct transmission to the auditory nerve through the bone and not through the medium of the drum-head and ossicular chain. I am at present carrying out some experiments in regard to this, on which I hope to report later.

APPENDIX.

T_0-T Seconds	I/I_0	I_0/I or hearing power	T_0-T	I/I_0	I_0/T or hearing power
2	2.294	0.436	9	41.9	0.0239
3	3.374	0.288	10	63.4	0.0158
1	1.514	0.660	12	145.6	0.00687
4	5.260	0.191	15	505.0	0.00198
5	7.970	0.125	20	4030.	0.00024
6	12.08	0.0828	25	32100.	0.00003
7	18.26	0.0548	30	255000.	0.000004
8	27.70	0.0361			

T_0 is time a normal ear can hear fork.

T is time a defective ear can hear fork.

I_0 is intensity of sound when normal ear ceases to hear it.

I is intensity of sound when defective ear ceases to hear it.

$I = I_1 \; e^{-KT}$

$I_0 = I_1 \; e^{-KT_0}$

K=0.415.

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November, 1913.

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Alterations in the Blood in Hay-fever. E. E. ROBERTS, *Brit. Med. Jour.*, May 30, 1914.

In hay-fever we have a decrease of the hemoglobins, a polycythemia, and an increase in the large mononuclears and eosinophiles. We also find vacuolated and degenerated leucocytes and some leucopenia. Asthma symptoms are accompanied by increased esinophilia. This blood picture in modified form persists in the intervals during the attacks. ED.

NEW TONSILLECTOME.

Dr. Harvey B. Searcy, Tuscaloosa, Ala.

The illustration shows a tonsil instrument that I had made in April, 1913, for use with the "Sluder technic."

In Dr. Sluder's instrument the fenestra made by the encroaching blade is crescent-shape and the middle of the convex edge of the blade engages the tonsil first, while the upper and lower part of the tonsil is not engaged until the tonsil is cut almost through. Occasionally part of the upper or lower tonsil tissue is left, necessitating more instrumentation. I have also found the hemorrhage to be too profuse.

The Beck-Mueller tonsillectome I have found better, but frequently the snare-wire gives trouble by breaking, slipping, or is

not properly adjusted in the groove made for it, and when not properly adjusted will be pushed ahead of the tonsil in operating and only a part of the tonsil removed. Then, too, when the tonsil is once grasped by the snare-wire the cutting has to be completed, as it is impossible to release the tonsil for a better placement, and a new wire is necessary for each tonsil to insure a smooth fit in the groove of the instrument.

I had a dull blade made to take the place of the wire in the Beck-Mueller instrument and find that it eliminates the above-mentioned disadvantages. The opening is always oval, engaging the whole tonsil, and with the dull blade and ecraseur a slow enucleation can be done with very little hemorrhage. By having the back of the sliding blade bevelled the tonsil is removed smoothly from the tonsil fossa while it hangs in the instrument and is removed from the mouth with it. By sliding the blade back to the original position the instrument is ready for the other tonsil, and where assistants are scarce the simplicity of adjustment saves considerable time and worry at a time when the operator is too busy to adjust instruments.

EDITORIAL DEPARTMENT

DEFECTS OF SPEECH.

EDITED BY
DR. G. HUDSON-MAKUEN,
PHILADELPHIA.

ON VOICE CULTURE.

One of the most notable of the recent books on voice culture is that from the pen of Mr. W. Warren Shaw, published by J. B. Lippincott Company, and entitled, "The lost vocal art."

Some of us may not know that the vocal art has been lost, but Mr. Shaw appears to make the fact very clear, and he tells us both when and how it was lost, and best of all how it may be regained.

According to Mr. Shaw, the vocal art began its wanderings when Garcia discovered the laryngoscope and taught us how to see the vocal mechanisms in operation. Up to this time, the old Italian masters, blissfully unconscious of the action of the vocal cords or of the various methods of breath-control, succeeded in developing and elaborating voices that have never since been equalled and that would put to shame the results attained by teachers of the present day. Strange to relate, these old masters were successful, not in spite of, but largely because of their ignorance of the physiological facts pertaining to voice culture, and their teaching began to deteriorate when they first saw the vocal cords in action and when, lured on by the little interesting knowledge thus obtained, they were led "like lambs to the slaughter" to study other physiological processes, such as the best methods of controlling the breath, thinking in their innocence that all this would make them better teachers and thus increase their usefulness. Evidence is given to show that Garcia himself, before he passed away, fully recognized the limitations of his discovery as applied to artistic voice-culture, and made but little use of the instrument in connection with his teaching.

The laryngoscope, according to Mr. Shaw, is an instrument par excellence for the physician, rather than for the singer. The

physician may safely and profitably study the physiological activities of the vocal mechanisms during voice-production, but for the singer to do so to any great extent can result only in disappointment and disaster. As a proof of this theory Mr. Shaw points to the failures resulting from the modern physiological schools of voice-culture, and he claims that there have been absolutely no successes. The vocal art so highly developed by the old masters has been practically lost because modern teachers have endeavored to supplant the older empirical methods of voice-culture with the newer physiological, and as they supposed, more scientific methods, forgetting the important fact that artistic singing is more largely a psychological process than it is a physiological one. Moreover, in the making of a singer, according to Mr. Shaw, and in the development even of hs peripheral mechanisms, psychological methods must predominate. In other words, it is always wrong for the singer to seek results by thinking primarily of the things that produce the results. This is the method of the physiological school which has resulted in so many diastrous failures.

The resurrection of the lost vocal art, Mr. Shaw tells us, must come through the idealization of tone and the striving for results directly, rather than indirectly through the physical mechanisms. The singer cannot successfully idealize a tone and think of the action of any muscle or group of muscles at the same time, and any attempt to do so is necessarily subversive of good results.

The normal, and therefore, the scientific, method of voice-production is first to think or idealize the tone, and the attempt to produce this idealized tone will tend to call up the requisite physical activities for its production.

The great singer, to be sure, is always largely endowed with a musical instinct and a musical mind, and these so-called psychological characteristics are of really greater value to him than his physical endowments; but on the other hand may we not be carrying the psychological theory too far when we aim to exclude from the singer a careful study of the physiological processes of his art?

It would seem that physiological methods must be used as an aid in the correction of defects of voice and speech, but when no marked abnormalities exist and when artistic results are to be attained in a comparatively and physiologically normal individual, psychological methods should be given precedence. As Mr.

Shaw points out, the best all-round physical development for the singer who is physically normal must come largely through his efforts to idealize tone, and to meet the ever-increasing demands of his own ear, made more and more exacting and critical through his work with the teacher.

To idealize tone simply means to call up an ideal tone in the special auditory tone center of the brain and to actually hear it in the imagination before it has issued from the throat. This is the normal physiological and psychological process of voice-production. The idealization of tone in this sense is what furnishes the stimuli necessary for the production of tone, and the more nearly perfect the idealization, the more nearly perfect will be the tone and the more uniform and harmonious will be the resultant physiological or neuro-muscular development.

The great singer, therefore, is the one who has learned to think and to idealize great song, and he is the one who has acquired the ability through long practice with an expert teacher to mentally and physically hear his own voice aright. He comes to be finally, then, his own critic and teacher. Physiologically, he is a highly developed organism, working under the immediate direction of a highly developed and artistic mind.

The keynote of Mr. Shaw's teaching, as outlined in his book, is the importance and absolute necessity of subordinating the physiological to the psychological in artistic singing. The singer must aim for results rather than for special physical activities, and his attention must be directed to the results to be attained rather than to the methods of attaining them. The physical activities required in artistic singing can better be aroused and developed through the idealization of results rather than through the method of directing the attention to the processes themselves.

Mr. Shaw is himself a good example as well as a good exponent of his psychological teachings, and he is more nearly right in respect to this phase of the subject than he is in respect to the physiological phase of it.

There are many who will not agree with him, for instance, when he says that in singing, the diaphragm is a muscle of inspiration and not a muscle of expiration; and physiologists or physicists will have difficulty in understanding how Mr. Shaw can make it possible for the diaphragm to contract strongly and the abdomen to retract at one and the same time, as he says they do during the more or less full inhalation preceding the tone-

production. Many of us also will fail to understand how it happens physiologically that in the full inhalation preceding tone-production, the abdomen is slightly drawn in, and in the emission of tone and necessarily also in the emission of some breath, the abdomen should be further retracted.

As Mr. Shaw says, Mendel was, of course, wrong in making the diaphragm an inspiratory muscle and advocating the protruding of the abdomen during a forced inhalation, but Mendel was at least consistent, because if the diaphragm is an inspiratory muscle, in full breathing the abdomen must protrude, because the diaphragm is the one muscle that can produce this particular phenomenon. . The fact is, as I have pointed out on various occasions, that the diaphragm is not an inspiratory muscle in the production of voice, and there is, as Mr. Shaw very properly says, a retraction of the abdomen in full inhalation; but the diaphragm is an expiratory muscle in the production of voice and it clearly comes into more or less action during the emission of the tone with a resultant protrusion of the abdominal walls.

A Quick and Effective Remedy for Otalgia. ADOLF SCHWARTZ, *Monatschr, f. Ohrenh.,* Heft. 3, 1914, p. 334.

The writer finds this remedy of advantage for pains in middle ear canal, in otalgia, tinnitus and when changing dressing. This wonderful "arcanum" has the poetical name of oleum sinapis aethereum. The writer, a military surgeon, had to travel all the way from the small village of Gravosa, not far from the scene of the Balkan war, to Vienna in order to prove at Prof. Urbantschitsch's Ear Clinic that the mere smell of oleum sinapis aethereum stopped ear pains in forty-four cases. The exact histories and symptomatology of the latter were given by the writer in a lecture before the Austrian Otological Society, February, 1914, and are published in the above article. GLOGAU.

BOOK REVIEWS.

Handbuch der speziellem Chirurgie des Ohres und der oberen Luftwege. Edited by Drs. L. Katz, H. Preysing and F. Blumenfeld. Band 2, Lieferung 2; Band 3, Lieferung 5-6. Verlag Curt Kabitzsch, Wuerzburg, 1914.

"Functional tests of hearing," by Dr. Gustav Bruehl: Recent editions on labyrinthine physiology and pathology add important chapters to this field. Prof. Bruehl covers this practical and vital fundament in his usual classic manner.

The method of using the Bezold tone-series and the differential diagnosis of this test are minutely described. The Galton whistle, Schaefer reasonators, Koenig cylinders, Struycken monochord, Gradenigo special fork, Hartmann diagrammatic tone fields, Barany noise apparatus are all given careful consideration. Nystagmus is presented in all its phases.

This is a splendid contribution and a most valuable reference monograph.

"The surgery of the external nose," by Prof. Otto Seifert is exhaustively presented and well illustrated and includes the latest form of therapy (Finsen light, radium, carbon dioxide snow, etc.).

The second monograph is on "Operations on the accessory sinuses of the nose," by Prof. Boenninghaus. Following an introductory chapter on differential diagnosis of inflammatory diseases of the accessory sinuses the author proceeds in a detailed description of the various operations for their relief.

These include in order: The various intra-nasal methods of opening the maxillary antrum; external methods through face via canine fossa; the facio-nasal method (Caldwell-Luc); modifications of the Gerber, Denker, Friedrich, Kretschmann, etc.

Then follows a detailed description of the technic of opening the frontal sinus both by the external and intra-nasal methods, and the various modifications suggested for this technic.

Operations on the ethmoid labyrinth are next considered and a concluding chapter treats of the operation on the sphenoid cells.

An independent chapter is devoted to a consideration of operations on the accessory sinuses not inflammatory nor suppurative in character, such as mucocele, cysts, polypi, osteoma and also the more radical surgical technic employed in removing malignant tumors of these areas.

Intra-cranial complications of extension from the accessory sinuses and their surgical treatment are also carefully considered.

This monograph is well illustrated with many original cuts.

The monograph on "Intra-nasal surgery on the nose," by Dr. L. Katz, first considers the various methods of rhinoscopy, and the important subject of intra-nasal hemorrhage, turbinectomy, removal of various types of polypi and other intra-nasal tumors, galvano-cautery, surgery of the turbinates, resection of the nasal septum and its numerous modifications, disposal of reflex neuroses in the nose, surgery of tumors of the nose both benign and malignant and their intra-nasal removal and gives an extensive contribution on the intra-nasal treatment of ozena, tuberculosis and endonasal syphilis, and a concluding paragraph on synechia, atresias and fractures of the nasal bones.

This monograph discusses this field of intra-nasal surgery minutely and includes a remarkably well-executed series of color plates and microscopic sections illustrative of the pathology of the parts discussed.

The significance and importance of "Orbital complications in nasal accessory sinus affections" is clearly defined in an exhaustive monograph by Prof. Hoffmann.

Much space is devoted to a careful description of the anatomy and topographical relations of the several sinuses to the orbit, to vascular and nerve distribution, and their relations to anomalies of the sinus in size, shape and position.

Special mention should be made of the splendid illustrations that accompany this monograph and the novel form of the series of stereoscopic plates to clinically illustrate the path of lesions of the orbit.

"The hypophysis, its pathology and operative treatment is the caption of the next monograph by Prof. A. Kuttner. The monograph is especially valuable for reference, as it describes the technic of the various types of operation undertaken to expose the hypophysis and remove tumors and pathological conditions of this tissue.

"The surgery of the tear ducts" is discussed by Gustav Ritter and is elaborated into a very formidable monograph. Appended to this is a bibliography of this subject-matter to date.

The concluding monograph of volume 3 is the "Surgical disposal of malignant neoplasms of the superior maxilla," by Dr. Heile.

While this field is not strictly to be considered within the scope of the rhino-laryngologist, it is nevertheless so closely identified with the surgery and pathology of our domain that it may be well given space in this important hand-book.

Each new monograph of this series as issued offers many surprises and excites greater admiration for this splendid monumental publication. There is nothing in the entire field of oto-rhino-laryngology that has ever been published which can approach it in importance, scientific strength, completeness and elaboration of publisher's art.

Development and Anatomy of the Nasal Accessory Sinuses in Man. Observations based on 290 lateral nasal walls, showing the various stages and types of development of the accessory sinus areas from the sixtieth day of fetal life to advanced maturity. DR. WARREN B. DAVIS, Corinna Borden Keen Research Fellow of Jefferson Medical College, etc., Philadelphia. Pp. 172, with 57 illustrations. W. B. Saunders Co., Philadelphia, 1914.

We are glad to see this evidence of independent research by an American colleague presented in an able volume entitled to take its place with the works of Onodi, Logan Turner and others.

Dr. Davis has collected and carefully studied large preparations of sections of the accessory sinus areas in the various stages of development from the early fetal to mature adult periods, special emphasis in this research being given to the study of specimens showing the conditions present during the period of childhood.

The difficulty of obtaining ample anatomical material for this purpose has been met by the development of a technic by the author by which the accessory areas could be removed *en masse* at the time of the post-mortem examination and still allow reconstruction of the face without marked disfigurement.

The total number of cases studied in the preparation of this work was 145, or 290 lateral nasal walls and accessory areas.

This monograph will, no doubt, be of considerable value in a careful study of accessory sinus anatomy and embryology and of much assistance in the further development of the field.

Operationen am Ohr. Die Operation bei Mittelohreiterungen und ihren intrakraniellen Komplikationen. Fuer Aerzte und Studierende. By DR. B. HEINE, Professor and Director of the K. Universitaets-Ohrenklinik und Ohrenpoliklinik, Munich. Pp. 232, with 29 illustrations in the text and 29 plates. Verlag, S. Karger, Berlin, 1913. Price, Mk. 8.80.

To readers familiar with the first edition of Heine's "Operations on the ear" this enlarged third edition will indicate the remarkable advance made in this field since the appearance of the first edition in 1903.

The operative technic of the labyrinth, semi-circular canal, dural and extra-dural abscess, and the disposal of the involved uvula bulb and vein in thrombosis and inflammation is here considered graphically and in original illustration.

Much emphasis is placed upon purulent affections of the middle ear traced through the oval and round windows, through the horizontal canal and involving the labyrinth, and the sinus, the character of pus invasions of the labyrinth, and the importance of radical labyrinthine surgery.

In citing the literature in this field the author seems to confine himself almost exclusively to German authorities.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, May 7, 1914.

DR. J. H. GUENTZER, CHAIRMAN.

SYNOPSIS OF DR. FREUDENTHAL'S PAPER.

(*Continued from page 764, August, 1914.*)

Deviated nasal septa or spurs, if they materially interfere with respiration, should be removed, and this can be done in almost any stage of tuberculosis. Adenoids should be curetted, but the tonsils should not be operated upon if the disease has been positively established. The writer had known of several fatalities which occurred in patients who had been sent away for recuperation and were operated upon contrary to his advice, dying of *pulmonary* hemorrhages in from three to eight days after the tonsillectomy.

When actual tuberculosis of the larynx has been established, the treatment of the condition is one of the most difficult that the laryngologist is confronted with, though it is well worth the work and the pains required, for the progress in the therapy of this condition has been marvelous. There is a great need for satisfactory sanatoria which will receive tubercular subjects with laryngeal complications. A prominent German scientist says that that hospital is the best which admits the worst cases, and with this statement the writer heartily coincides. The general treatment of the incipient or early cases often in itself effects a curative effect. The rest-cure, especially of *the voice*, the good air, and suitable nourishment, often result in the healing of slight lesions, superficial erosions, infiltrations, etc.

A more active therapy is indicated when there are ulcers, papillary outgrowths, localized and diffuse infiltrations. No matter what the conditions of the lungs, two requirements must be met,—the cough must be stopped and the dysphagia removed. The methods of accomplishing this must depend upon the experience and judgment of the physician. In some instances major operations are proposed, in others, partial or total laryngectomy. Theoretically, this is the correct method of procedure, but practically it has not proved a success. The majority of patients operated on in this way succumb quickly to pneumonia or other complications, and only a few recover from it. Even simple tracheotomy, which has been recommended as a prophylactic measure is of little value. It should only be performed on account of dyspnea which cannot otherwise be relieved.

One is therefore limited to minor surgical measures within the larynx. It is well known that the disease manifests itself frequently on the anteroposterior wall and on the anterior wall, i. e., the epiglottis. Deep ulerations and infiltrations may persist for a long time on the lateral wall without causing dysphagia or other serious trouble, but difficulties arise as soon as the arytenoids or the epiglottis become involved, and these conditions must be overcome or the patient is lost.

In severe infiltrations of the epiglottis, all efforts toward alleviating a pronounced dysphagia will fail on account of the inability to reach the ulcers low down in the larynx, the epiglottis occluding the introitus laryngis, or because they have formed on the laryngeal surface of the flattened epiglottis and cannot be reached by any application. Under these circumstances, the best that can be done is to amputate the epiglottis, either with some cutting forceps or with the cold snare. This operation is simple and enables one to treat the lesions lower down. Occasionally, when the epiglottis is the only part affected, improvement is noticed immediately. When ulcers are present lower down, they have to be especially treated.

In the treament of ulcerations, the first thing to strive for is to remove the pain on deglutition and incidentally relieve the laryngeal cough. To accomplish this, one must produce local analgesia of long duration,—painlessness of the parts lasting for several days. This is secured by the employment of orthoform, or anesthesia, applied as a powder or in an emulsion, the writer's formula for the latter being: Orthoform, 6,00; menthol, 0.5-5,00; formin, 0,50; ol. amyg. dul., 15,00; pulv. acaciae, 10,00; aquae ad, 60,00 M. f. emulsio.

The important points to be remembered in applying the emulsion or the powder are that (1) the parts to be treated must be clean and (2) that drugs must be applied in such a manner that they will remain on the ulcer. If too much is applied at one time it will be coughed up. A drop that remains on the spot and is absorbed will do more good than a pint that is coughed up. Such medication alone, if used correctly, will often produce cicatrization of an ulcer.

In case the ulcers do not heal under analgesic treatment, resort must be had to caustics,—chlorid of zinc, nitrite of silver, argyrol, etc., or the galvano-cautery, followed within half an hour by the insufflation of orthoform in powder or by emulsion. In deep, solitary ulcers, the galvano-cautery occasionally yields good results, but for multiple ulcers it cannot be recommended. The reaction after the galvano-cautery is sometimes unpleasant and unavoidable. Fulguration has given some encouraging results.

Suspension laryngoscopy has opened up a new field for therapeutic endeavors. With it, the larynx is opened widely to direct inspection and an excellent opportunity is afforded to reach its different parts, especially the posterior wall which is so frequently affected. One can also curette thoroughly all ulcerations, which, after all, is the most satisfactory surgical method. The writer has done this frequently with excellent immediate results, although the ulcers recur and have to be curetted over and over again. The procedure is not painful and the dangers connected with it are practically nil. There is, however, danger of edema of the larynx, and for this reason it is often wise to proceed slowly and to curette only some of the most troublesome parts at the first sitting. After doing curettage under suspension laryngoscopy, it is best to apply a strong caustic, such as the galvano-cautery or pure lactic acid.

Any necessary excisions of infiltrated portions or of granulations can best be accomplished under suspension laryngoscopy. The writer has been amazed at the ease with which inter-arytenoid papillary out-growths can be removed by this method.

Very helpful are the different rays used therapeutically during recent years,—the actinic rays, the high frequency current, and the rays emanating from radium. One of the first orders given to a patient is that he should keep in the sun as much as possible, in the winter. In the summer the early morning hours are recommended, up to nine or ten o'clock. As a substitute for the sun, the electric arc light can be used and its chemical or actinic rays applied externally to the affected parts.

In cases in which none of the therapeutic measures indicated avail, injections of alcohol have sometimes given relief. This was first suggested by Rudolph Hoffman, whose aim was to produce analgesia of long duration. He recommends an 85 per cent alcohol at a temperature of 45°C or 112°F. After the subsidence of the initial pain, which is sometimes quite severe, another injection is immediately made. Hoffman uses a strong, but somewhat blunted needle, as it avoids the blood vessels and is less liable to break.

The treatment and supervision of laryngeal tuberculosis in private practice is difficult and sometimes impossible, but in the milder cases much can be accomplished.

A successful practitioner ought to be able to cure at least 50 per cent of his patients afflicted with tuberculosis of the larynx in any stage of the disease. Optimism is nowhere so much indicated as in the therapy of tuberculosis, for it greatly assists the physician as well as the patient.

DISCUSSION.

DR. CARTER thanked Dr. Freudenthal for bringing this very important subject before the section, for while we may not introduce anything very new in the way of diagnosis, prognosis, or treatment, still we have opportunity to emphasize those points which have been found most useful in treating this protean disease.

As to the question of climate, which is the first thing that occurs in the matter of treatment, it is generally conceded that a fairly cold climate of moderate altitude and fairly free from dust is the ideal climate for a laryngeal case. Such cases do not stand dust of any kind; it keeps up the cough and makes it very much worse. All treatment must be directed against the cough, for the cure of tuberculous laryngitis is obtained after all, not so much by the treatment as by the rest. The constant coughing and hacking keeps up the irritation, and no rest is obtained. Therefore, the best method of controlling the cough must be considered. The cough in laryngeal cases is almost always annoying. When the inter-arytenoid sulcus is much involved the slightest thing will start the cough. Then, the pulmonary condition has much to do with the cough. We must find out the controlling element and direct our treatment to that. Prevention is the most important point of all in the treatment of tuberculous laryngitis. There is nearly always a catarrhal condition in the tuberculous cases, and this catarrh should be carefully attended to.

There are no positive early signs of laryngeal tuberculosis. For that reason, one should pay particular attention to the condition of the upper air passages, especially a catarrhal condition of the larynx.

Dr. Carter said that the question of the cough recalled his early experiences on the staff at Bellevue Hospital, when he saw many of these laryngeal tuberculosis cases. They were very interesting, and the patients were kept in the wards as long as possible, for the purpose of studying them. The method adopted for controlling the cough and diminishing the laryngeal irritability was very effective. It consisted in the use of a zinc inhaler on which was sprinkled 10 drops of a mixture of beechwood, creosote, chloroform and alcohol. This was used every hour or two during the day, and frequently at night. It certainly diminished the coughing, and in that way helped to cure the laryngeal tuberculosis,—or at least it ameliorated the suffering of the patients, for most of them were advanced cases.

As to the local treatment, Dr. Carter said that he had tried almost everything that had come under his observation. He could not exactly agree with Dr. Freudenthal regarding the use of powder in laryngeal tuberculosis, for it seemed to irritate the larynx and cause more coughing. Powder does not have any local effect until it has become a solution with the secretion in the larynx. Most of the powders used are insoluble or slowly soluble, and do not remain in the larynx long enough to have any effect. The treatment which he has found most useful is formaldehyd, two to four per cent, applied after cleaning off the parts with cotton pledgets. This is somewhat irritating, and sometimes it is necessary to use cocain before applying it.

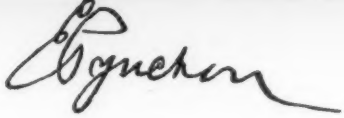
Dr. Carter said that he does not approve of the use of cocain in laryngeal tuberculosis cases any more than is absolutely necessary, for there is always a certain amount of edema following its use, and stronger and stronger solutions have to be used.

Operative procedures should be limited to those which are of the sthenic type, especially those cases where the tubercular formations are circumscribed and in the curettage of old indolent ulcers on an indurated base, and in unilateral tuberculosis, that will allow the removal of as much tissue as possible and leave one side of the larynx in good shape.

Dr. Yankauer said that several years ago he had showed to the members of the Section an apparatus which he had devised which enabled the patient to medicate his own larynx. This was done at the suggestion of an internist, for the purpose of applying solutions and emulsions to the larynx in tuberculous cases, so that they could nourish themselves better. It consisted of a tube bent at right angles at one end and fitted to the patient's mouth with a small flange of adhesive plaster, so that when the patient introduced it into his mouth, the flange rested against the teeth, and when the fluid was injected it would reach the larynx. This device has been used a good deal since that time, and it was no doubt familiar to most of those present.

He had been trying to use a modification of this device which has succeeded well, and had been much encouraged to continue this work. He had used it with a patient who had a marked tuberculosis of the epiglottis; half of the epiglottis had disappeared, and the ulceration extended along the side of the tongue almost to the front of the mouth. Most of the ulcerations were healed and cicatrized when the patient died of other complications.

(*To be continued.*)



IN MEMORIAM.

Dr. Edward Pynchon died at his home in Chicago, August 28th, in the sixty-second year of his life, of uremia, after a brief illness.

Dr. Pynchon was a graduate of the Eclectic Medical Institute, Cincinnati, 1873; of the Medical College of Ohio, Cincinnati, 1876; a member of the American Academy of Ophthalmology and Oto-Laryngology; and professor of rhinology, laryngology and otology in the Chicago Eye, Ear, Nose and Throat College.

In the death of Dr. Pynchon THE LARYNGOSCOPE loses one of its most valuable and ardent collaborators and the oto-laryngological profession an active, earnest and indefatigable worker.

His mechanical ingenuity was a recognized feature of every national oto-laryngological meeting at which his models of new instruments and apparatus were demonstrated.

He was a practitioner of the old school who depended largely on practical observation and common sense in the pursuit of his chosen field.

He was a frequent contributor to oto-laryngological literature and among his more important contributions we mention: "The bête noir of the vocalist," "The degenerate tonsil," "Directions for the control of nasal hemorrhage," "Nasal bougies and drainage tubes," "New mechanical saw for intra-nasal operations," "New nasal speculum," "New nebulizing device," "Pneumatic massage in aural practice," "Surgical correction of deformities of the nasal septum," "Technic of tympanic inflation," "Tonsillectomy by electro-cautery dissection," and "Tonsillectomy in children under general anesthesia—a hospital operation."

We shall miss his genial companionship, his practical counsel, his stimulating energy and his loyal support as a friend and co-worker in practice and medical journalism.

We extend our sympathies to his bereaved family.